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Overview of the Flaccid Hemiplegic Shoulder following a Stroke

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OVERVIEW OF THE FLACCID HEMIPLEGIC SHOULDER FOLLOWING A STROKE

by

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This Independent Study, submitted by Tricia R. Pederson in partial fulfillment of the requirements for the Degree of Master of Physical Therapy from the University of North Dakota, has been read by the Faculty Preceptor, Advisor, and Chairperson of Physical Therapy under whom the work has been done and is hereby approved.

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PERMISSION

Title	Overview of the Flaccid Hemiplegic Shoulder Following a Stroke
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Signature Lucia R. Pederson

Date March 21, 1995

TABLE OF CONTENTS

LIST OF FIGURES

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ABSTRACT

Stroke may cause long-term disability and impairment in the hemiplegic shoulder. Problems may develop early in the flaccid phase of recovery and complicate the entire rehabilitation process. The etiology and treatment of shoulder complications remains uncertain. Contributing factors include inappropriate handling and positioning, abnormal muscle tone, impaired sensation, and musculoskeletal pathology.

Prevention and treatment of shoulder dysfunction begins with positioning, early range of motion, and education. Physical therapists play an important role in stroke management by administering therapeutic positioning programs, teaching appropriate exercise regimens, and instructing in proper handling techniques

The purpose of this paper is to review methods being utilized in the clinic to rehabilitate the flaccid shoulder and prevent post-stroke complications that may interfere with mobility and function. Understanding the possible causes is essential for choosing appropriate prevention and treatment strategies. Future work needs to be done in this area as the painful shoulder continues to be a serious problem in stroke hemiplegia.

CHAPTER ONE

INTRODUCTION

Stroke, medically referred to as a cerebrovascular accident (CVA), is the third leading cause of death and the major cause of adult disability in the United States.[1-3] Approximately 500,000 Americans experience a new or recurrent stroke each year. Nearly 30 percent of the people die within the first thirty days following a stroke. For those that survive, 30 to 40 percent will suffer some degree of permanent disability requiring long-term rehabilitation or nursing home care. The remaining stroke survivors will sustain mild residual impairment. Some of these patients return home without too much difficulty and some even resume their careers. In all, roughly 70 percent of stroke victims survive the central nervous system damage and must learn to live with subsequent mild to severe neurological deficits.

The incidence of stroke rises rapidly as age increases, with the majority of stroke patients ranging between the ages of 65-74 years.[2] Epidemiological studies show that the incidence of stroke has been declining over the past several years due to the identification of risk factors, education, treatment, and increased awareness for prudent living.[1] However, the prevalence of stroke victims living in this country appears to be rising due to an enhanced survival rate and a growing elderly population. Currently, there are 3 million people living in the United States with stroke after-effects. Health care professionals need to understand the resulting disability following a stroke, since more people are surviving and seeking medical consult for rehabilitation.

A stroke occurs when there is a restricted flow of blood supply to the brain, producing cell damage and impaired neurological function.[2,3] Possible causes for CVA include thrombosis, embolism, hemorrhage, and spasms within the blood vessel walls.[4] Generally, symptomatology and recovery of function are dependent upon several factors including: 1) the location of ischemic process; 2) the size of cerebral impact; 3) the functional structures involved; and 4) the availability of collateral blood flow.[2-5]

Clinically, a variety of deficits are possible including impairments of sensory, motor, cognitive, perceptual, and/or language functions.[2] Motor impairments appear to be the most common and disabling. Motor deficits usually occur unilaterally and on the opposite side of the body as the cerebral hemisphere traumatized by vascular occlusion. Hemiplegia in the afflicted extremities and trunk commonly result from a CVA, making it the most classic and obvious sign of neurovascular disease of the brain.[3] The term hemiplegia is often used to refer to both muscle weakness (sometimes called hemiparesis) and paralysis.

Patients who have suffered a stroke with resulting hemiplegia often develop shoulder disability and/or pain because of pathologic changes that occur in the shoulder complex after a CVA.[6] Physical therapy is regularly requested in order to aid in the motor recovery of the upper extremity and prevent painful complications. Preventing shoulder dysfunction and pain are important goals for the rehabilitation team for two main reasons.

Blue = 1st stage (post-CVA)

First, the stability and mobility provided by the shoulder dictates what the remainder of the limb can do for environmental manipulation; therefore, promoting functional movement of the shoulder is essential for improving quality of life.[7] Second, shoulder complications can interfere with the rehabilitation program if pain is limiting active patient participation. A painful upper extremity can disturb sleep, cause depression, require added medications, or cause the patients to withdraw from family and/or medical support, making the rehabilitation of hemiplegic patients extremely challenging.[8,9]

Immediately following a stroke, flaccidity commonly develops in the involved extremities. The flaccid state occurs in approximately 90 percent of the patients and it may last for hours, days, or weeks depending on the severity of cerebral damage.[7,9,10] This stage of recovery is characterized by a lack of muscle tone, voluntary muscle action, and deep tendon reflexes.[11] Normally, the muscles surrounding the shoulder complex offer the most stability for the glenohumeral joint. After a stroke, the muscles are unable to provide the necessary support for the shoulder so distracting forces usually act upon the ligaments and joint capsule.[10-14] These soft tissue structures offer very little shoulder protection and as a result, the ligaments and capsule often overstretch, resulting in glenohumeral subluxation and/or pain. During this stage, the shoulder is susceptible to injury since the muscular support is not available to counteract gravitational forces. Moreover, the change in tone of the shoulder musculature frequently results in an alteration of the normal orientation and biomechanics of the scapula and humerus, predisposing the glenohumeral joint to further subluxation. Consequently, the shoulder is often involved in a variety of post-stroke complications which has shown to prolong the course of rehabilitation and limit functional recovery.[7]

Inferior glenohumeral subluxation and pain are two of the most troublesome complications interfering with restorative efforts provided by therapy services.[2,5,9,10,13,14-21] Subluxation and pain can occur together or they may occur separately. The association between them remains unclear. Many clinicians consider the pain to be the result of subluxation, by virtue of traction on the joint capsule and rotator cuff musculature.[11] However, the exact mechanism of pain has yet to be established since there are several cases when shoulder subluxation of varying degrees is not painful; in as much, there are many painful shoulders with no significant subluxation.

Shoulder subluxation can be a debilitating problem; therefore, it has been the subject of numerous studies.[10,12-14,16,21] The majority of clinicians and researchers agree that subluxation initially occurs during the flaccid stage of recovery. If neglected, it can persist into the spastic stage of recovery as well. Here, it may be more painful than in the flaccid shoulder. Van Ouwenaller and associates[18] identified a much higher incidence of shoulder pain in spastic than in flaccid hemiplegia, 85 percent to 18 percent respectively.

Researchers and clinicians are unsure of the etiology of hemiplegic shoulder pain.[22,] The literature presents numerous factors which may cause this phenomenon. Besides glenohumeral subluxation, other musculoskeletal conditions include rotator cuff tears[7,18,] brachial plexus injuries, impingement syndromes, reflex sympathetic dystrophy, degenerative changes[5] ,and abnormal muscle tone.[7]

Mechanical factors causing pain have also been noted in the literature. One recurring suggestion found in the literature and clinical practice is that poor handling and positioning of the affected arm may traumatize the shoulder joint.[5] Exercising the involved extremity inappropriately has been found to lead to shoulder pain as well.[23]

Since the arm is used for so many functional activities, investigators have performed many studies using stroke patients as subjects; the objective being, to find the most optimal ways of restoring function to the shoulder. Various treatment methods have been devised and advocated but the issue still remains controversial.[3] For instance, the use of shoulder slings or supports has received both praise and criticism in reducing shoulder subluxations.

Even though the goals in physical therapy are to maintain normal joint alignment and reduce subluxation and/or pain, management of the hemiplegic shoulder continues to be unsettled. Evidence is insufficient to support the various causes of shoulder complications during central nervous system recovery.[5] Few studies have clearly documented the effectiveness of treatment techniques.[24]

Choosing appropriate treatment methods for the flaccid hemiplegic shoulder demands a thorough understanding of the anatomy and biomechanics of both the normal and pathologic shoulder, the progression of neurological disease entity; the possible causes for shoulder pathology, and finally the rationale supporting the use of different treatment techniques. The purpose of this literature review is to discuss current methods being used to rehabilitate the flaccid shoulder in order to reduce post-CVA complications that would negatively interfere with mobility and function.

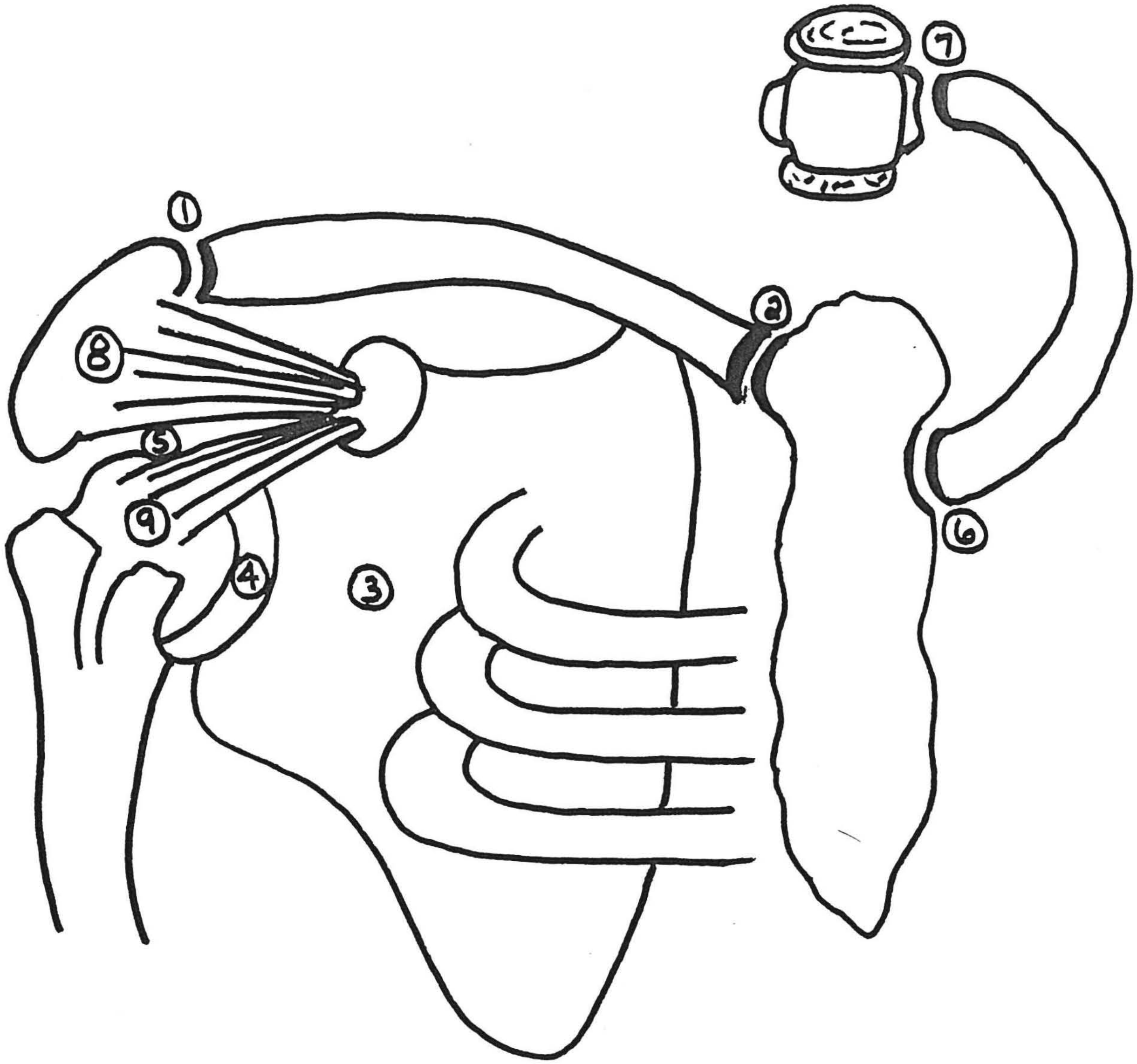
This paper will include a review of the anatomy and physiology of the flaccid hemiplegic shoulder, a discussion of various shoulder complications and potential reasons for their development, and finally, a presentation of post-stroke rehabilitation techniques. The paper will emphasize the importance of correct handling and positioning of the upper extremity, as well as the use of supportive devices during the flaccid stage of recovery.

CHAPTER 2

SHOULDER JOINT ANATOMY AND BIOMECHANICS

When describing the shoulder, most authors discuss the acromioclavicular (AC), the sternoclavicular (SC), the scapulothoracic (ST), and the glenohumeral (GH) joints.[25,26] Cailliet[4,11] expands this list to include the suprahumeral, costosternal, and costovertebral joints. (Fig. 1) Most authors agree the shoulder joint is better termed the shoulder girdle complex because it is a composite of many articulations that act in harmony to place the arm and hand in functional positions. All joints of the shoulder complex move synchronously and each articulation depends on the others to insure full and painfree range of motion.[26,27] Together, these articulations provide the shoulder with a range of motion that surpasses any other joint in the body. Impairment of any one of these joints causes a disruption in normal movement and results in shoulder dysfunction.[6]

The normal shoulder can move through almost a complete arc of motion in both the sagittal and frontal planes, which allows the arm and hand to assume various positions in space for environmental manipulation.[25] Because of this wide range of mobility, people use their arms to perform many athletic endeavors and activities of daily living. The greatest proportion of shoulder motion occurs at the GH joint and this is the articulation most often involved in post-stroke shoulder complications.[4,26] As a result, stroke patients commonly lose the mobility of the shoulder as well as the functional capability of



1. Acromioclavicular joint
2. Sternoclavicular joint
3. Scapulothoracic joint
4. Glenohumeral joint
5. Suprahumeral joint
6. Costosternal joint
7. Costovertebral joint
8, Coracoacromial ligament
9. Coracohumeral ligament

Figure 1. Shoulder girdle complex.

the entire upper extremity. Limitations in the GH joint will eventually cause restrictions in other joints. Dysfunction will occur since the natural rhythm of movement between the scapula and humerus will be destroyed. Losing control of the arm can be very devastating for stroke survivors.

GLENOHUMERAL JOINT

The GH joint consists of a synovial articulation between the convex humeral head and the concave glenoid fossa.[26] Authors agree that the humeral head points in a superior, medial, and posterior direction. However, the direction in which the glenoid fossa faces in the normal resting position of the scapula continues to be controversial. According to Basmajian, Bazant, and Cailliet,[8] the fossa faces upward as well as anteriorly and laterally. In contrast, Prevost et al.[12] speculate that the glenoid cavity inclines in a downward rather than upward direction. The anatomical configuration of the GH joint allows for significant range of motion; however, this mobility makes the joint very unstable and vulnerable to injury. Unlike the ball-and-socket joint at the hip (which acquires its stability from the rigid bony union formed between the acetabular fossa and femoral head), the GH joint relies heavily on the stability created by surrounding soft tissues.[26]

The rotator cuff muscles--supraspinatus, infraspinatus, teres minor, and subscapularis--provide the majority of the support for the GH joint.[26] These muscles are referred to as the dynamic stabilizers of the joint because they guard against anterior, posterior, and inferior displacement of the humeral head from the glenoid fossa with humeral elevation. Because of their important contributions to stability, the rotator cuff muscles are given the term "guardians of the shoulder."[8] The long head tendon of the biceps brachii contributes to the dynamic stability of the GH joint as well.[27]

The static stabilizers of the joint include the GH ligaments, the coracohumeral ligament, the joint capsule, and the glenoid labrum.[26,27] The GH ligaments--superior, middle, and inferior-- reinforce the joint capsule anteriorly, while the coracohumeral ligament supports it superiorly. When the arm is adducted or in a dependent position, the superior GH ligament aids in the prevention of inferior displacement of the humeral head. The middle and inferior GH ligaments serve to prevent anterior dislocation or subluxation of the humeral head, especially during the upper ranges of motion. Like the superior GH ligament, the coracohumeral ligament provides stability for the dependent arm by resisting the downward pull of gravity on the humeral head.

The joint capsule attaches medially to the glenoid fossa and laterally to the anatomical neck of the humerus. (Fig. 2) Because it is so thin, it offers very little primary support except in the superior region of the joint.[7] The surrounding ligaments and rotator cuff muscles help to strengthen the capsule. The fibrous glenoid labrum lines the perimeter of the fossa.[26,27] The glenoid labrum is lined internally by a synovial membrane; externally, it attaches to the joint capsule. The labrum contributes to GH joint stability by deepening the shallow glenoid fossa to provide better congruency between the socket and humeral head. Even with the labrum, the glenoid fossa is much smaller than the humeral head. In fact, the surface area of the fossa in only one-third to one-fourth that of the

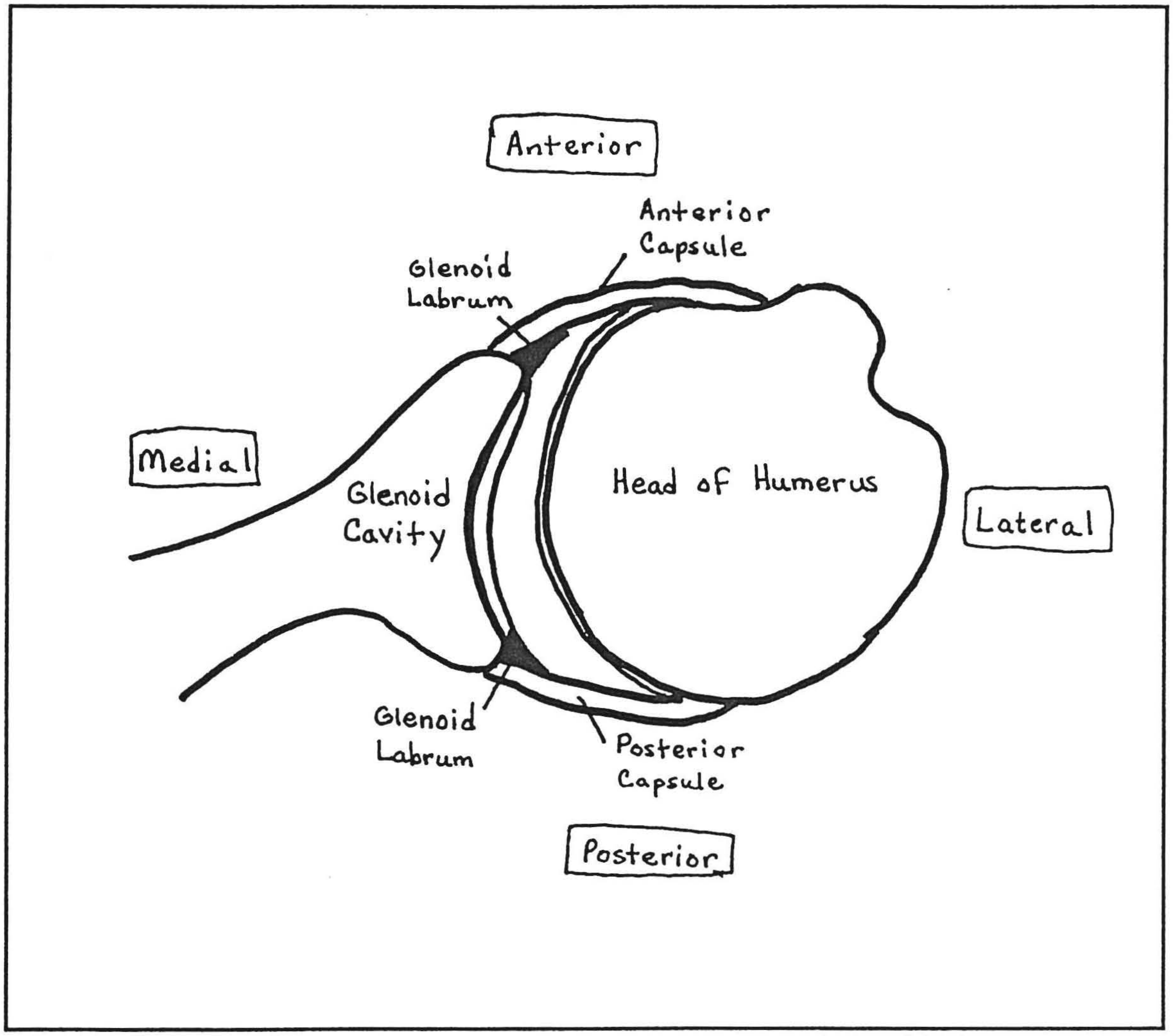


Figure 2. Horizontal view of the glenohumeral joint capsule and glenoid labrum.

humeral head, which means that only 25-30 percent of the head is in contact with the glenoid cavity at any given time. As a result, the GH articulation is relatively unstable.

SCAPULOHUMERAL RHYTHM

In order to attain full and painfree motion of the upper extremity, scapular rotation through movement at the SC and AC joints, must accompany humeral elevation.[26,27] The scapula normally rotates upward and outward with shoulder elevation. This motion is accomplished through the action of the serratus anterior and trapezius muscles. Together, these muscles act as a force couple to rotate the scapula. Scapular rotation insures that the glenoid fossa remains in an upward tilt to provide a base for the humeral head to move. It contributes 60 degrees to the available 180 degrees of average shoulder range of motion. In stroke hemiplegia, a loss of muscle tone, gravitational influences, imbalanced patterns of motor return, and postural abnormalities may alter the normal operation of the scapulothoracic mechanism.[25]

CORACOACROMIAL ARCH

The coracoacromial ligament unites the coracoid and acromion processes of the scapula and forms the coracoacromial arch, or the protective roof overlying the GH joint.[27] This arch is sometimes referred to as the suprahumeral joint (a false joint) because it helps to prevent trauma from above as well as superior dislocations. The rotator cuff tendons, biceps tendon, joint capsule, and subacromial bursa occupy the subacromial space between the humeral head and the coracoacromial arch. The bursa separates the deltoid muscle and coracoacromial arch that lay above it from the supraspinatus and biceps tendons that lay below it. It allows for smooth gliding between these structures and reduces the friction on the tendons as they pass under the arch with humeral elevation. In individuals with no pathology of the shoulder, the distance measured radiographically between the acromion process and the humeral head averages 9-10 mm. Supraspinatus and bicep tendon tears are likely to occur when this space is reduced. Inflammation of the tendons or bursa will cause a reduction in the subacromial space and lead to impingement syndromes, resulting in pain and restricted motion.

In stroke hemiplegia, impingement syndromes and rotator cuff tears are possible and may lead to a painful shoulder. Improper positioning and trauma by mishandling the upper extremity or GH subluxation are probable causes for the presence of these disorders.

BRACHIAL PLEXUS

The nerve supply to the entire upper extremity originates from a complex network of neural tissue called the brachial plexus. The anterior rami of cervical roots 5 through 8 and thoracic root 1 (formed from a dorsal sensory and ventral motor root) join together to form the plexus.[28] Anatomically, the anterior rami lie between the scalene muscles of the neck and run beside the subclavian artery. The plexus continues distally and passes over the first rib. The five anterior rami unite just above the clavicle to from the upper trunk

(C5 and C6), the middle trunk (C7), and the lower trunk (C8 and T1). Each trunk divides into an anterior and posterior division which gives rise to the lateral, posterior, and medial cords. Many branches emanate from the cords, trunks, and roots to supply the muscles of the shoulder, scapula, and distal upper extremity muscles.

Hemiplegic stroke patients have the potential to develop traction or pressure injuries that involve the neural structures of the brachial plexus.[29,30] Common sites of injury include the C5 and C6 roots, long thoracic nerve, axillary nerve, suprascapular nerve, and others. Proposed mechanisms include prolonged lying on the hemiplegic arm and subluxation of the flaccid shoulder. Extensive lying on the paralytic upper limb may cause either a pressure or traction injury to the brachial plexus. Subluxation of the shoulder generally delivers a traction stress to the neural tissues of the brachial plexus. Inappropriate or lack of support for the paralyzed arm and improper handling of the flaccid arm during transfers are probable causes to the subluxation and subsequent brachial plexus injury.[31] A brachial plexus injury is a serious post-stroke complication that will interfere with rehabilitation efforts substantially. Nerves regenerate at a very slow rate, approximately 1 mm per day. As a result of this slow process, motor recovery will be delayed.

PATHOANATOMY AND PATHOMECHANICS

Following the onset of a CVA, patients usually experience a paralysis of the affected side, which is marked by a period of initial muscle flaccidity or low tone, followed by a gradual or sudden reflex increase in muscle tone and function.[32] It is uncertain how long patients remain in the flaccid stage before progression is made into subsequent stages. It appears to vary among stroke survivors, lasting only a few hours or days in some patients or extending into weeks or months in others. A duration lasting longer than two weeks has been considered prognostically unfavorable. This areflexic state is characterized by a reduction in active postural tone, a loss of motor control, and an absence of reflex activity in the head, neck, trunk, and extremities.[25]

Gravitational forces dramatically influence the body during this acute phase as well, and the effects of gravity can be detrimental to the shoulder. Patients have no power to contract the muscles that move the upper extremity in the flaccid stage. Not only have the patients lost all kinetic function, but also, they have lost all static control to support the arm against gravity.[11] The patients generally adopt a posture of lateral flexion in the head and trunk toward the hemiplegic side. The righting reflexes of the vertebral musculature lose their postural support to keep the spine erect against gravity. With this functional scoliosis towards the hemiplegic side, the scapula becomes depressed and rotated downward. In this position, the angle of the glenoid fossa faces downward instead of upward. This may be a precipitating factor in the inferior descent of the humeral head away from the glenoid cavity of the scapula. Muscle shortening on the involved side will be inevitable if the patient is allowed to remain in this position for extended periods of time. Without proper muscle length, mobility of the scapula and shoulder will be compromised, thus functional use of the involved extremity limited.

The loss of motor control and postural tone in hemiplegia disrupt the normal anatomical and biomechanical relationship between the scapula and humerus. Other factors preventing normal shoulder mechanics include the development of abnormal movement patterns, secondary soft tissue changes that block motion, and GH subluxation.[25] During the initial flaccid state, patients cannot move, but as motor return occurs, individual muscles gradually increase in tone. Various muscles attaching to the scapula are often the first to regain tone. The rhomboids, latissimus dorsi, pectorals, and levator scapula normally function to rotate the scapula downward. As motor return occurs in hemiplegia, early patterns of control are typically unbalanced. When this happens, the muscles encircling the joint do not return at the same time or strength. Extensor control usually dominates over flexor control. This early pattern of motor return forces the scapula and humerus into abnormal postures. Eventually the normal alignment between the scapula and humerus changes because certain muscle groups are positioned in their shortened ranges while opposing muscle groups are in their lengthened ranges. This activity enhances spasticity because the shortened muscle fibers have the potential to be in a continuous state of contraction. Consequently, the arm may still appear flaccid even though tone has increased in the scapular muscles.[8]

During the flaccid stage, the scapula loses its stability on the thorax. The pull of gravity on the arm and trunk, postural asymmetry, and early patterns of motor return greatly influence the position of the scapula.[25] Left untreated, the changes occurring in the soft tissue structures may cause the scapula to become immobile on the thorax, which will ultimately interfere with normal scapulohumeral rhythm by blocking both scapular and GH motion. The serratus anterior and upper trapezius cannot function to counteract the contractile forces of the downward rotators as the arm elevates. Pain-free and full GH range of motion will thus be prevented.

Glenohumeral subluxation occurs after a stroke when the stability of the shoulder joint and shoulder girdle is compromised.[14] Investigators have conducted many studies on inferior subluxation of the GH joint in stroke patients, but none have confirmed the actual etiology for its frequent appearance. One explanation is the presence of downward forces pulling on the arm, such as gravity, improper positioning, or mishandling during transitional activities. In the absence of muscle tone, the distracting forces may cause the ligaments, joint capsule, and musculature to stretch, especially the supraspinatus, which is important in maintaining good alignment of the humeral head within the glenoid.[10,12]

Basmajian and Bazant,[8,12,20] and Cailliet[11] have proposed a mechanism for inferior joint subluxation that has gained considerable recognition over the years. They suggested that the normal orientation of the scapula on the thorax allows the glenoid fossa to face superiorly, anteriorly, and laterally. The upward slope of the fossa helps to prevent the humeral head from sliding down the fossa. In order to sublux inferiorly, the head would need to move laterally. When the arm is adducted, the superior portion of the capsule and the coracohumeral ligament are taut; however, in abduction the superior capsule becomes lax and is unable to provide the necessary support. Muscles of the rotator cuff must contract to provide the GH joint with adequate stability. In summary, this "locking" mechanism does not function with the arm in abduction.

Patients with flaccid hemiplegia have lost this passive "locking" mechanism of the shoulder because the scapular muscles, particularly the serratus anterior and upper trapezius, have lost their ability to keep the scapula angled upward.[8,11] According to Cailliet,[12] the scapula depresses and the humerus assumes an abducted position with the arm held by the side. Because the capsule has more slack with the shoulder in abduction, the head of the humerus slides down the fossa, resulting in an inferior subluxation. Normally, the combined actions of the serratus anterior and upper trapezius muscles keep the scapula from medially rotating in a non-hemiplegic limb. With the glenoid fossa facing downwards, the inferior angle of the scapula migrates closer to the vertebrae and appears lower than the scapula on the opposite side. If scapular winging is noted on the affected side, muscle tone has generally increased in certain muscle groups to pull the medial border of the scapula away from the ribs. Basmajian and Cailliet[12] also propose that inferior subluxation could be a result of the relative abduction of the humerus seen in flaccid stroke patients. In regards to treatment, preserving the upward angle of the glenoid fossa has been an important goal to prevent shoulder dislocation in hemiplegia.

These theories have not gone unchallenged, however. Prevost et al.[12] conducted a study using hemiplegic stroke patients to investigate the relationship between the relative angle of humeral abduction and the degree of subluxation. From their results, they concluded that the orientation of the scapula and the position of the humerus are not important factors for the occurrence of GH subluxation. To support this claim, they found that the glenoid fossae of both the involved and uninvolved sides face downward. More importantly, the glenoid fossa on the affected side had a less downward slant than on the unaffected side. This means that the glenoid fossa on the normal side is angled more in a downward direction than on the hemiplegic side. Other authors have also noted similar results in their studies using normal subjects.[12] Therefore, Basmajian's model of the glenoid fossa sloping upward should be questioned, and it can be assumed that the normal angle of the fossa is rotated in a downward direction. Provost et al.[12] also challenged Cailliet's suggestion of a depressed scapula with shoulder girdle flaccidity and/or spasticity. It appeared to them that with rotational movements, the scapula adopted more of an upward angle. Furthermore, it was concluded that the abduction of the humerus, relative to the scapula, cannot be considered a factor in the development of shoulder subluxation.

Shai et al.[13] proclaimed that the presence of a radiologic sign, which consisted of a V-shaped opening between the glenoid cavity and humeral head, may help in early diagnosing of shoulder pathology following stroke. If recognized early, it is believed that orthotic intervention may still be helpful in preventing GH joint subluxation. In their study, 12 out of 14 patients that showed this sign went on to develop chronic painful shoulders and 4 of them developed inferior subluxation. They go on to explain how the V-shaped sign is formed. The inferior glenoid labrum is the first structure to resist the caudal drift of the humeral head in shoulder subluxation. The labrum acts as a fulcrum as the humeral head impinges upon the inferior glenoid cavity. The caudal pull of gravity on the unsupported arm causes the humeral head to rotate about this fulcrum. As a result, the shaft of the humerus adducts and the humeral head abducts from the glenoid cavity to form a V-shaped opening in the upper portion of the fossa. In later stages, the humeral head migrates caudally into a subluxed or dislocated position. (Fig. 3)

Arsenault et al.[33] conducted a follow up study on the clinical significance of the V-shaped space in hemiplegic patients. They too found that the V-shaped space was significant between the nonaffected and affected shoulders for the subluxed group. However, this was only evident on a 45 degree radiographic view and not a 0 degree or frontal plane view. Their results also indicated that an inferior GH subluxation may occur without any downward rotation of the scapula or abduction of the humerus, which according to Basmajian and Cailliet, accompany subluxations. Lastly, they discovered there was no significant relationship between subluxation and later development of shoulder pain, which contradicted the findings found in the study by Shai et al.[13]

The muscle fibers of the supraspinatus, infraspinatus, and posterior deltoid run horizontally. This allows them to provide the most support in preventing GH subluxation.[8] The coracohumeral ligament reinforces the superior portion of the capsule to resist inferior migration of the humeral head as well. These structures counteract the forces of gravity to simultaneously shift the head laterally and inferiorly. Chaco and Wolf[14,20] reported that the integrity of the supraspinatus is a contributing factor in GH joint subluxation. In their investigation, they found that subluxation was present in patients

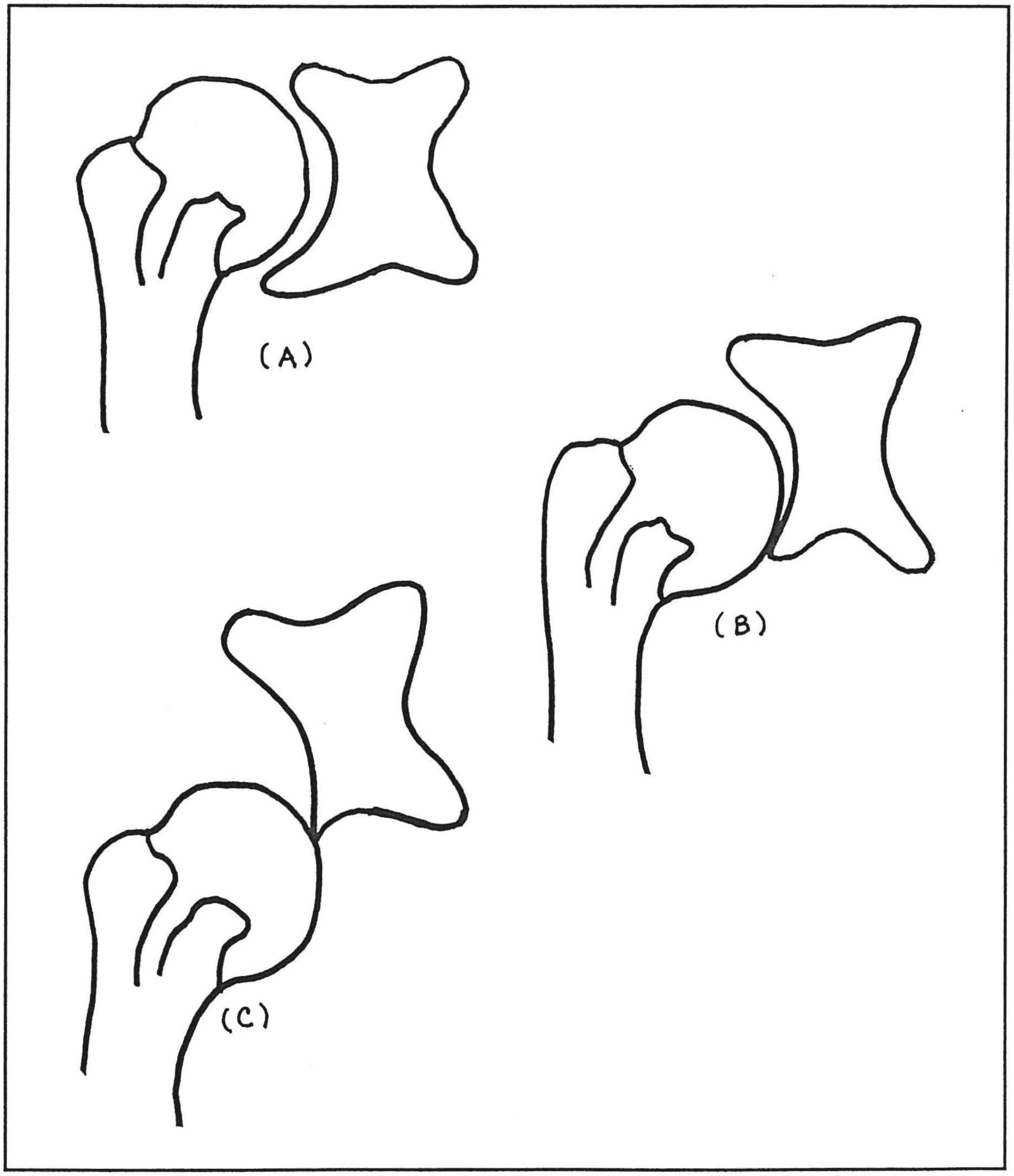


Figure 3. A.) Alignment of the glenohumeral joint in a normal shoulder. B.) Early development of a V-shaped space between the humeral head and glenoid fossa of a hemiplegic shoulder. C.) Subluxation of the glenohumeral joint in the hemiplegic shoulder.

whose flaccid supraspinatus did not show any electromyographic activity with the arm dependent or loaded. They advised that loading of the arm be avoided in order to elude a stretch on the soft tissues.

CHAPTER THREE

POST-STROKE SHOULDER COMPLICATIONS

Flaccidity results immediately after a stroke when the excitatory center of the central nervous system is damaged.[4] In the early stages of hemiplegia, patients generally lose all contact with the flail upper extremity. No movement can be initiated and sensation is usually absent. Full passive range of motion is possible but there is no resistance felt at any time during the movement. Because the shoulder loses its supportive mechanisms during the flaccid stage, it is a site for many post-stroke complications. Examples of shoulder problems commonly experienced by stroke survivors include GH subluxations, pain, impingement syndromes, rotator cuff tears, traction neuropathies, and shoulder-hand syndrome. The onset of such secondary complications may "prolong the course of rehabilitation and limit functional outcome."[7]

For years, the hemiplegic shoulder has been the subject of many studies. However, much of the information in the literature that pertains to the characterization, prevalence, diagnosis, etiology, and treatment of shoulder pathology remains controversial, despite efforts that have been allocated to stroke rehabilitation through investigative studies.[7] Several reasons for this conflict exist. First, strokes are infinitely variable in the site of a lesion and the degree of extensive brain damage. Due to brain plasticity, the same lesion may create both similarities and differences among stroke victims. Second, people recover from central nervous system damage differently. The literature suggests that the greatest recovery occurs within the first three to six months following a CVA; however, functional gains are still possible one to two years later. Function may emerge, change, or plateau at any given time and as a result, researchers have a difficult time in selecting a homogenous stroke population to study. Third, investigators study parameters of shoulder pathology that are difficult to objectify, such as pain and tone. Large variances in the process of gathering data may yield inconsistent study results.

Shoulder pathology may occur directly from neurologic impairment found in stroke hemiplegia.[4] Disturbances in motor control may lead to immobility and impaired movement mechanisms.[7] Fluctuations in muscle tone may cause flaccidity or spasticity in varying degrees. Abnormalities in sensation may enhance the perception of pain or do just the opposite, cause a loss of pain and proprioception, which have the potential to create other problems.

Shoulder complications seen in hemiplegia may also result from unrelated disease pathology.[4,5,7] Considering that stroke usually affects the elderly population, problems may directly arise from degenerative changes occurring in the shoulder. Preexisting conditions of the shoulder joint that were latent before a stroke may be enhanced by the hemiplegia. And finally, trauma to the shoulder joint may be the result of mishandling, improper positioning, or falls sustained during a CVA. This chapter intends to highlight some of the common hemiplegic shoulder complications that may lead to disability in stroke survivors.

GLENOHUMERAL SUBLUXATION

Glenohumeral subluxation occurs when any of the structures contributing to joint stability are disrupted.[6,14,25] The exact etiology of how shoulder subluxation develops is unresolved. Perhaps the most important factor is the position of the scapula on the thorax. With the onset of muscle paralysis, the scapula begins to slope in a downward direction. As a result of this malalignment, the humeral head slides down the glenoid cavity as a result of traction forces. Flaccidity of the rotator cuff musculature, especially the supraspinatus, may be the cause for shoulder subluxation. Rotator cuff tears or bicipital tendon tears may play an influential role in causing subluxation.[20,29] Finally, subluxation may result from brachial plexus involvement.

The incidence of GH subluxation varies substantially from 17 percent[20] to 92 percent[14] in stroke survivors. Inferior subluxation occurs more frequently than any other type of subluxation and it appears to develop primarily in the flaccid phase of recovery.[3,20] Shoulder subluxation seems to occur less frequently in the spastic stages or as motor function returns. Once the subluxation has occurred however, evidence suggests it is irreversible, despite the return of reflexive tone or voluntary muscle action.

Diagnosis of the subluxed shoulder is made by using oblique x-ray films with the patient in a vertical posture and the arm in a dependent position.[17] Clinical and radiographic methods are currently available to measure the degree of subluxation. Clinical methods lack precision and are tester dependent because they entail using palpatory skills or small instruments to measure the suprahumeral space. Radiological methods are either qualitative or quantitative. Qualitative techniques involve visual inspection of x-rays followed by classification of the subluxation into a predetermined number of categories. This method lacks precision as well because the categories of subluxation are limited and they may be subject to interrater variability. Quantitative measurements have the advantage of being more precise than any other method but they have the disadvantage of exposing the patients to harmful levels of radiation. One type of quantitative measurement compares the anteroposterior (AP) X-rays obtained from the involved shoulder and uninvolved shoulder. With this type of measurement, two or more X-rays are required. The second type measures the amount of subluxation of the affected shoulder by using the plane of the scapula method. The plane of the scapula method was designed to overcome problems with existing techniques. With this method, a precise measurement of both vertical and horizontal subluxation can be obtained with a single X-ray in a specific plane.

Pain may or may not accompany a subluxed shoulder and their association is unclear. Tobis and other authors speculate that inferior subluxation is the cause of post-stroke shoulder pain.[7] In one study, 26 out of 32 stroke patients had some degree of malalignment and of these 26, 25 complained of pain.[20] Shai et al.[13] claims that stroke patients who show an early sign of subluxation will more than likely go on to develop shoulder pain. In their study, 12 out of 14 clients who displayed this sign went on to develop pain. These results are convincing of a strong association between subluxation and pain; however, there are studies and observations that have indicated no significant relationship between subluxation and pain. For example, the Bobaths concluded that shoulder subluxation is not responsible for shoulder pain, but it is the improper handling of the subluxed shoulder which causes the pain.[34] The findings in the study by Van Langenberghe and Hogan[20] revealed three important points. First, no statistical difference was found in the degree of pain between patients with and without subluxations. Second, no correlation existed between grade of subluxation and degree of pain, which is contrary to previous notions that greater subluxations caused more pain. Finally, the authors suggested that subluxation was not a significant factor in the development of shoulder pain. It has been said that "subluxation is not painful as long as the scapula [remains] mobile."[25]

THE PAINFUL SHOULDER

A painful shoulder presents a serious problem for the patient, family, and entire rehabilitation team. Patients commonly demonstrate a sequence of pain and a vicious cycle generally ensues.[8] Patients who have pain with movement will keep the arm immobile. Those who have pain at rest will not participate in any form of active rehabilitation program. The patients become focused on their pain, lose their concentration, and are easily distracted from learning new skills. Regaining functional independence is difficult because the pain and stiffness interfere with ADLs. Balance reactions are disturbed, making sitting, standing, and walking impossible tasks. The patients become depressed, lose self-esteem, and withdraw from family and/or social support. The patients are unable to cooperate in therapy sessions because the pain has kept them awake all night. As a result, the patients make very slow progress and become discouraged, anxious, or frustrated.

The incidence of shoulder pain in hemiplegia ranges from 5 percent to 84 percent.[19] The causes of shoulder pain have not been clearly identified.[34] Because the shoulder is comprised of so many complex structures, it is very unlikely that a single mechanism exists to cause the shoulder pain experienced by stroke survivors. A painful hemiplegic shoulder has been found to occur with muscle tone imbalances, a loss of joint ROM, improper handling or positioning, impingement syndromes, tendinitis, and other musculoskeletal problems.[25]

According to Ryerson and Levit[25], there are four categories of shoulder pain: 1) joint pain, 2) muscle pain, 3) pain from altered sensation, and 4) shoulder-hand pain syndrome. Joint pain develops in the shoulder when the GH joint is held in an improperly aligned position as a result of muscle imbalances or improper movement patterns. This occurs when there is an insufficient amount of rotation of the humerus with elevation or if the humeral head is aligned incorrectly within the glenoid cavity. With shoulder joint pain, patients generally complain of discomfort that is "sharp" and "stabbing" while performing passive or active movements either in a weight-bearing or non weight-bearing position. In order to relieve this type of pain, the movement must stop immediately and the bones of the joint must be realigned.

Muscle pain occurs when a shortened muscle is lengthened too quickly or stretched beyond the range of comfort, usually when performing upper extremity weight-bearing activities.[25] Patients can localize this type of pain and they typically describe it as a "pulling" sensation. The pain is relieved when the stretch ceases or is decreased a few degrees. Decreasing the stretch will allow the shortened muscle to lengthen without feeling the discomfort.

Pain due to altered sensitivity from CNS impairment occurs during the acute stage of recovery.[25] This type of pain can be diffuse and aching and/or localized and sharp. It is commonly felt during a treatment session that has required tactile, sensory, kinesthetic, or proprioceptive stimuli. If it develops, the treatment should stop for that session and monitored continuously in subsequent sessions to avoid sensory intolerance.

The shoulder-hand syndrome (SHS) is believed to be a variant of the disorder, reflex sympathetic dystrophy.[7,29] It is a very debilitating post-stroke complication that interferes with the overall rehabilitation process. This neurovascular disorder is characterized by varying degrees of pain, stiffness, edema, trophic skin changes, and vasomotor instability in the upper extremity.[4,7,8,29,31,34] If the syndrome is allowed to progress, atrophy of the skin, muscle, and bone will take place. The condition culminates with the presence of soft tissue deformities and joint contractures involving the hand and fingers. The fixed and permanent deformities will ultimately limit the functional use of the hand in the future.

Diagnosis of SHS is clinical, yet it can be very difficult because stroke patients without SHS can suffer similar symptoms.[7] The clinical course of SHS varies but it can be divided into three stages according to the severity of symptoms.[8,34] The disorder begins with an aching[25] or burning[7] sensation throughout the upper extremity. The hand suddenly becomes edematous and is limited in ROM. The edema is most prominent on the dorsum of the hand and five digits. The creases in the skin over the metacarpophalangeal (MCP) and interphalangeal joints (PIP and DIP) diminish. The edema elevates the extensor tendons and prevents flexion and terminal extension of the joints. With the MCP joints in extension, the collateral ligaments never elongate.[4] They shorten and prevent further flexion of the fingers. Shoulder ROM is limited and it may or may not be painful. Finger abduction, supination and wrist extension are painful and are also limited in motion. The color of the hand changes to a pink or lilac hue and the skin becomes shiny, warm, and either dry or moist, and the nails become opaque compared to the opposite hand. The patients often become hypersensitive to touch, pressure, and movement. This beginning stage may last anywhere from 3 weeks to 6 months depending on intervention.

The second stage lasts 3 to 6 months and is characterized by increasing severity of symptoms, except for the edema which begins to subside.[7] The pain becomes unbearable to any type of pressure and a bony prominence forms on the dorsal aspect of the intercarpal-metacarpal junction.[8] Osteoporotic changes become evident on X-ray as well.

The third and final stage is marked by hand and finger deformities and a permanent loss of mobility.[7,8,34] The hand adopts a posture that resembles an intrinsic minus or an intrinsic plus hand.[4] With the first malformation, the wrist is fixed in flexion and ulnar deviation, the MCPs in extension, and the IPs in flexion through tenodesis action of the flexors. With the second type of deformity, the MCPs remain in flexion and the IPs in extension. The condition is usually not painful and the edema disappears completely by this stage. "The edema, containing protein, converts into a diffuse cobweb-like scar tissue that adheres to the tendons and joint capsules and prevents further movement. The joints undergo disuse atrophy of the cartilage with thickening of the capsule."[4]

The incidence of SHS in stroke patients with hemiplegia is uncertain.[31] Its occurrence has varied among studies, ranging from 0 to 50 percent. Studies have found that SHS develops less frequently in stroke victims with mild impairment and more frequently in patients with severe motor involvement. SHS has been observed in both the flaccid and spastic stages of recovery, 1 to 5 months[7] after a stroke. Patients with this syndrome have been noted to have increased confusion, have greater sensory losses, have a past medical history of cardiac problems, and have higher incidences of GH subluxations.

The cause of SHS has not been proven. Cailliet[4] suspects that this syndrome develops when the major circulatory pumps of the hand or axilla become impaired. Immobilization of the upper extremity, especially in a dependent position, and continued sling use may initiate the disorder. Prolonged flexion of the wrist under pressure, overstretching of the joints in the hand, intravenous infiltrates, and accidents to the hand are examples of other possible causes promoting edema in the hand.[8]

Prevention begins with avoiding the factors that precipitate SHS.[8] Fortunately, most of the hazardous mechanisms can be avoided by educating the hospital personnel, patient, and family. Positioning with supportive devices at the wrist is an effective prevention strategy. Weight-bearing activities and ROM exercises are very important and should not be avoided. A slight hand position change or controlled movements during weight-bearing tasks can reduce the pressure at the wrist. Availability of ROM should be established by the therapist by first checking the uninvolved hand. And lastly, if at all possible, intravenous lines should not be placed in the hemiplegic arm.

The most important step in the beginning stages of SHS is early intervention.[7,8,34] Treatment consists of proper positioning, avoidance of wrist flexion by using cock-up splints, tables, or wheelchair supports, centripetal wrapping, and ice baths. The use of modalities, such as transcutaneous electrical stimulation, contrast baths, and paraffin is controversial. Active range of motion exercises are preferred over passive exercises whenever possible to get the "pumping" action going by muscular contraction. Oral corticosteroids have proven to be very successful in conjunction with physical therapy treatments. Surgery may be indicated if conservative measures fail. Symptoms lasting longer than six months without effective treatment has a dismal outlook.

IMPINGEMENT SYNDROMES

The incidence of hemiplegic shoulder pain associated with impingement is unknown.[7] Abnormal contact between the humerus and structures of the coracoacromial arch may lead to impingement syndromes of the musculotendinous cuff in a hemiplegic shoulder. The supraspinatus tendon, subacromial bursae, and bicipital tendon are commonly involved. In stroke patients, impingement of soft-tissue structures may occur during passive range of motion exercises by inadequate rotation of the humerus and scapula.[2,7,31] Repeatedly failing to provide upward rotation of the scapula and external rotation of the humerus with abduction will traumatize the soft structures and eventually lead to inflammation, degeneration, and pain. Upward rotation of the scapula elevates the coracoacromial arch and external rotation of the humerus alters the position of the greater tubercle in relation to the arch. Other predisposing factors for an impingement disorder include downward rotation of the scapula and inappropriate support positioning. Downward rotation of the scapula increases the relative abduction of the arm. If the arm is left unsupported, the coracoacromial arch assumes a lowered position and an earlier impingement is felt during abduction exercises. Improper supportive devices for the upper extremity, especially at the elbow, may force the humeral head into overlying structures and cause an impingement. Therefore, prevention begins with avoiding these mannerisms.

The typical symptoms seen with an impingement in stroke hemiplegia are similar to other musculoskeletal disorders, unrelated to hemiplegia.[7] Abduction exacerbates the symptoms, especially within the painful arc between 60 and 120 degrees. There is tenderness noted upon palpation of the humeral head with the arm in extension and humeral rotation. Atrophy of the rotator cuff may be seen. Magnetic resonance imaging (MRI) and pain relief with subacromial anesthetic injections are helpful in establishing the diagnosis.

Therapeutic measures consist of discovering the source of impingement; using modalities and nonsteroidal anti-inflammatory drugs for reduction of pain and inflammation; decreasing tone in the scapular depressors; and modifying an exercise program in the functional ranges of motion.[7] If conservative measures do not relieve the problem within 6 months, an acromioplasty may be needed.

ROTATOR CUFF TEARS

Superior disruption of the rotator cuff may result from impingement, ischemia, or trauma.[7] Chronic impingement may lead to rotator cuff tears (RCT). RCT have been associated with impingement during passive abduction exercises beyond 90 degrees without lateral rotation.[31] The blood supply to the supraspinatus is diminished when the flaccid extremity is left unsupported in a dependent position. As a result, ischemia develops within the "critical zone" of the supraspinatus tendon, making it more susceptible to tears. Falls which occur at the onset of hemiplegia may result in traumatic RCT. Traction injuries and premorbid degenerative changes may also facilitate cuff pathology in hemiplegia.

Tears may be partial or full thickness tears. Arthrography or MRI can confirm the diagnosis. Symptoms will be similar to those of an impingement syndrome. Tears may be painful or painless. If there is pain, it may increase at night and positioning is unable to relieve it. Conservative approaches are indicated initially for treatment, but if they fail, surgery is then the treatment of choice.

TRACTION NEUROPATHIES

Stroke patients have the potential to develop complications associated with a brachial plexus injury (BPI) or proximal mononeuropathy (PMN).[30] These injuries are generally a source of shoulder pain and upper extremity dysfunction. Possible mechanisms for injury include lying on the hemiplegic shoulder for extended periods of time,[30] inappropriate handling of the flail upper limb during transfers or dressings,[29] improper positioning and lack of support in bed or in a wheelchair, and subluxation of the GH joint.[31] Nerve damage may occur from either a traction or a compression injury. The patients who are unconscious during the acute stage are at an increased risk of suffering from traction or pressure neuropathies. Common sites of involvement include the C5-C6 nerve root,[29] long thoracic, median, and ulnar nerves, upper portion of the brachial plexus,[30] radial, axillary, suprascapular, and musculocutaneous nerves.

Health care providers should suspect a BPI when patients demonstrate an atypical pattern of motor recovery than what is expected.[4,29,31] Clinical manifestations of BPI or peripheral nerve injuries include 1) fair to good distal return and intrinsic hand function with poor return of proximal musculature, 2) segmental muscle atrophy, 3) extension contractures of the fingers, 4) delayed onset of spasticity, and 5) electromyogram (EMG) abnormalities. In hemiplegia, proximal motor return normally occurs before distal return, where in a BPI, this sequence is reversed. Flaccidity in the flail hemiplegic limb may be difficult to differentiate from the flaccid limb with lower motor neuron (LMN) involvement. Atrophy will not be as marked in hemiplegia as it would be with peripheral nerve injuries. Deviation from the typical flexor pattern of the hemiplegic upper extremity, especially with extension contractures, should signal a nerve problem. And finally, abnormal EMG readings are not as evident in stroke hemiplegia since it is a test for LMN involvement. Diffuse EMG abnormalities and delayed nerve conduction latencies have been noted in the hemiplegic upper extremity, although, these findings reflect more of motor axon deterioration secondary to the cortical lesion rather than a BPI or peripheral nerve injury.

Diagnosis of a BPI or PMN can not be based entirely on EMG abnormalities. In fact, the physical examination is the cornerstone in detecting nerve injuries. Other signs of peripheral nerve involvement besides atrophy and flaccidity include weakness, anesthesia, altered sensation, burning pain, abnormal temperature regulation, color changes and decreased muscle stretch reflexes localized in a specific myotomal or peripheral nerve distribution.

The incidence of BPI is uncertain.[7] In the study by Meredith et al.,[35] 5 out of the 12 patients were suspected as having pathology of the upper portion of the brachial plexus. Prognosis depends on the site and extent of lesion.[7] Proximal upper trunk lesions have a better prognosis than lower trunk lesions. BPI hinder the recovery of the upper limb and they may impede rehabilitation for 8-12 months since nerve regeneration takes place at a very slow rate, approximately 1 millimeter per day. Prevention focuses on positioning and supporting the arm with supportive devices, such as slings, lap boards, arm troughs, pillows, or foam wedges, throughout all sedentary and mobile activities. Treatment consists of gentle ROM exercises with sensory cues, medications, and modalities.

CHAPTER FOUR

POSITIONING & HANDLING OF THE HEMIPLEGIC SHOULDER

Acute stroke rehabilitation may begin once the medical condition of the patients has stabilized. Generally, this is within the first 72 hours following a stroke.[2] Early physical therapy intervention will help facilitate awareness, initiate patient participation, improve mobility, and prevent secondary complications. Besides engaging in therapy services, what the stroke patients do during the remaining hours of the day and night will determine the success of the rehabilitation program.[8] Studies indicate that stroke patients in hospital or rehabilitation settings spend 30-50 percent of the daytime in passive pursuits such as lying in bed, sitting unoccupied, or watching television.[32] Assuming postures that promote spasticity or other secondary complications will interfere with restorative efforts.

An effective program also depends on what the medical team and family do when caring for the patients. Improper handling of the hemiplegic upper extremity during times not at therapy is believed by some to cause shoulder pain and/or subluxation.[8] Gains made during therapy sessions will either be lost or they will not carry over into daily life. Therefore, primary objectives during early stroke rehabilitation are to enhance proper positioning of the patients in all postures and promote effective handling techniques during activities continuously performed during the inpatient stay. Physical therapists can insure their rehabilitation efforts will be restored 24 hours a day if they educate the patients, the family, and the medical staff involved with patient care responsibilities.[31] Following the positioning and handling guidelines established by the therapists early on in a rehabilitation program can help prevent secondary complications, which could ultimately limit future active movement and participation by the patients. Rehabilitation emphasizes a 24-hour management and should be regarded as a way of life.[8]

Muscle imbalances and changes in muscle tone or posture make the shoulder joint extremely vulnerable to injury during the flaccid stages of recovery.[34] Trauma to the shoulder often occurs during range of motion (ROM) exercises and functional activities like rolling, scooting, sitting up, transfers, lying in bed, and positioning readjustments. Because the shoulder and scapular muscles have no voluntary control or contractile ability in the flaccid state, they are unable to provide the support needed to protect the GH joint during these events completed throughout the day.[10]

The adverse effects of inappropriate positioning and handling are great. Positioning the involved arm in undesirable postures may lead to spasticity and contractures. The onset of this involuntary muscular activity may impair the patient's potential for using or regaining control of the affected upper limb.[32] Several therapeutic principles to stroke rehabilitation have been advocated by the Bobaths, Rood, and Brunnstrom. Although the concepts of their techniques differ in emphasis, they all recognize the importance of reducing spasticity. It is widely agreed that positioning out of the typical spastic patterns will discourage the development of post-stroke spasticity.

Therefore, encouraging the patient to assume, and the caregivers to use "reflex-inhibiting" postures is vital to any positioning program.

Past investigators have hypothesized that subluxation does not result from incorrect handling techniques; rather, it occurs spontaneously during the early stages following a stroke when the patients begin to engage in sitting and standing activities.[8] Researchers have based this assumption on test results obtained from their subjects, who were hemiplegic patients with total paralysis. All of the patients showed a malalignment of the shoulder on X-ray when in a seated position during the first three weeks following a CVA. Since then, it has been the general consensus that shoulder trauma can be avoided by proper positioning and handling skills.[34]

In the early phases, stroke patients spend the majority of the day in bed so positioning is an important adjunct to therapy.[8] Paying attention to all the components of the shoulder complex when positioning is essential; failing to do so may hinder the patients' abilities to regain function or cause a painful upper extremity.[11] Positioning programs must assure that the cervical and thoracic spines, the scapula, and the GH joint are correctly aligned in all postures. Programs must also consider gravity and how it influences the flail extremity, as well as the position of the whole body. Undesirable positions include: shoulder girdle retraction and depression; adduction and internal rotation of the humerus; elbow flexion and forearm pronation; ulnar deviation and flexion of the wrist and fingers; and thumb adduction.[4,11] Positioning in these positions will enhance spasticity and the development of flexor synergy patterns.

According to the literature, the shoulder should be protracted with the arm brought forward when lying on the affected side, lying on the unaffected side, lying supine, sitting up in bed, and sitting in a chair.[32] Placing the scapula in a protracted and upwardly rotated position will protect the shoulder by preventing a downward displacement of the humerus.

Two areas of controversy evolve when positioning the upper extremity.[32] The first is the number and height of pillows (or table) to use when supporting the shoulder and arm in a forward position. Some authors feel that the upper arm should rest on a pillow when lying on the involved side while others do not. The second area of controversy concerns the method for positioning the arm out of an adducted and internally rotated posture. Some authors indicate that the shoulder be placed in external rotation in the supine position, while others imply that the shoulder be in external rotation and a slight degree of abduction. For chair sitting, some authors note the importance of bringing the arm forward on a resting surface, while others advise abduction and external rotation of the shoulder. Dardier[32] pointed out the advantages of utilizing both positions when sitting. First, slight shoulder abduction and external rotation help to support the trunk, thereby holding the forearm in the preferred supinated position. Second, placing the arm onto a table in front of the patient keeps it in the visual field and facilitates bilateral integration of the hands. Abduction and external rotation allow the pectoralis major to be stretched, which will lead to a reduction in resting tone and an increase in its extensibility. If not positioned in abduction or external rotation, patients will assume an internally rotated and adducted arm position. Prolonged positioning in this posture will lead to hypoextensibility and muscle shortening by sarcomere resorption.

Research supports the significance of proper upper limb positioning. Scheduling position changes every 2-3 hours should help to stimulate the patient and provide adequate pressure relief.[2,8] However, if beginning signs of pressure sores become evident, rotating between positions must be done more frequently to prevent ulcer formation.

LYING ON THE AFFECTED SIDE

Contrary to previous notions that pressure should be avoided upon the involved upper extremity, it is allowed and beneficial, but only if the scapula is protracted and the humerus is flexed, ideally to 90 degrees, to eliminate direct pressure on the shoulder joint.[8,11] Lying on the affected side is the most important position of all and it should be introduced as soon as possible. The benefits of this position include a reduction in spasticity, an elongation and increased awareness of the hemiplegic side. In addition, patients prefer this position over others and it allows the uninvolved hand to continue with functional activities.

The head should be well supported and kept in a slightly flexed position. Cailliet[4,11] suggests that the head be flexed laterally and rotated toward the unaffected side to inhibit the typical synergy pattern of neck flexion to the hemiplegic side. O'Sullivan[2] recommends that the trunk be straight, whereas Davies[8] advises the trunk to be rotated slightly backwards and supported by a pillow from behind. The hemiplegic shoulder is drawn forwards and flexed at an angle not less than 90 degrees. The therapist assisting the patient can reach under the thorax to bring the scapula forward into protraction. The body weight of the patient allows him or her to remain in this position. This should be a comfortable position. If the patients complain of pain or discomfort, it usually means there is not enough protraction. Distally, the forearm is supinated and the wrist is passively dorsiflexed.

The unaffected arm should be placed in an appropriate position also. Recommendations for positioning the uninvolved limb are similar for all positions except for lying on the afflicted side.[32] A few authors favor forward placement of the arm, some prefer resting it on top of the body, and others stress a backward placement. Davies[8] recommends either resting the arm on the body or on a pillow behind the back. Placing the arm forward will cause the trunk to come forward, resulting in scapular retraction on the involved side. Even if the patients have intravenous lines in the arm, it is still extremely important to turn them on a regular basis as well.

LYING ON THE UNAFFECTED SIDE

The only differences noted when lying on the sound side occur in the trunk and the hemiplegic arm positions. Again, the head is well-supported. The trunk is neither in a semi-prone or semi-supine position, but it is to be at right angles with the surface of the bed.[2,8] The involved shoulder should be protracted and elevated to approximately 100 degrees with the arm resting on a pillow. The purpose of the pillow is to prevent the arm from crossing the chest into horizontal adduction, which could exert a traction force on the suprascapular nerve and cause shoulder pain.[31] A small pillow may be used underneath the rib cage to allow elongation of the hemiplegic side.

LYING SUPINE

The supine position should be minimized as much as possible and should be balanced with other positions, since the patients will be more susceptible to pressure sore development.[2,4,8,11] Supine lying also maximizes abnormal reflex and extensor activity due to the influences of the tonic neck and labyrinthine reflexes. However, supine lying may be needed for those patients who do not tolerate sidelying.

In supine, the head and trunk should be in midline or slightly flexed toward the sound side, once again for elongation purposes. A small pillow or towel roll under the affected scapula will assist in scapular protraction. Another pillow under the arm will insure an optimal position of elevation, elbow extension, forearm supination, and passive wrist dorsiflexion. If unable to maintain the shoulder position using pillows Bohannon et al.[15] have described a shoulder positioning device made of foam and Velcro to hold the arm in both abduction and external rotation to prevent internal rotation of the arm in the splint. An alternative position for the involved arm is to place it above the head in extension.[4,8,11]

SITTING IN BED

Sitting up in bed should try to be avoided as much as possible since the upright posture is difficult to maintain in bed.[2,8] Flexion of the trunk is encouraged when the head of the bed is elevated. Totally eliminating this posture is not feasible for the stroke patient who must sit up to eat or drink. When sitting up in bed, pillows should be placed behind the patient to keep the spine erect as much as possible. The head should be left unsupported so it facilitates the cervical extensors to hold the head up. The arms may be supported on a pillow or adjustable table lying across the bed to encourage scapular protraction and weight-bearing or approximation to the GH joint.

SITTING IN A CHAIR

Getting out of bed is indicated as soon as possible in order to avoid the serious complications associated with prolonged bed immobilization, such as pressure sores, thrombosis, and respiratory difficulties.[8] A vertically orientated position provides stimulation and it demands more of an active role from the patients. Sitting in a chair allows better posturing than what can be achieved sitting up in bed; therefore, it is advisable to transfer patients from the bed to the chair periodically throughout the day. Once again, the arms can be supported on a lab board or table to allow for scapular protraction and bilateral integration.

HANDLING TECHNIQUES

Correctly handling the hemiplegic limb during ROM and functional activities is just as important as providing the arm with a good resting position. The shoulder girdle complex reacts adversely to immobilization because of its intricate construction.[8] Degenerative conditions may result from prolonged immobilization and give rise to shoulder pain.[5] "Exercise is the most important therapy in restoration of physical independence in hemiplegia."[23] Passive exercises started early in the rehabilitation program will serve to maintain normal motion in the flaccid limb and preserve normal integrity of the joint capsule.[2] Utilizing correct ROM techniques will protect the vulnerable state of the shoulder. Studies done in the past have noted that passive abduction of the humerus can lead to rotator cuff injuries with subsequent pain.[23] When elevating the arm, special attention should be given to external rotation of the arm with scapular mobilization and upward rotation. Adequate external rotation is needed for safe abduction of the shoulder beyond 90 degrees.[15] Patients are likely to experience shoulder impingement, rotator cuff injury, and/or pain with inadequate movement of the scapula during humeral elevation. Encourage active control of the extremity by asking the patients to hold the arm at various positions throughout the arc of movement.[4,11]

Therapists must educate the patients on self-mobilization exercises in all planar motions utilizing the above techniques. Before elevation of the arm is attempted by the patients, therapists should first show them how to clasp the hands together by interlacing the fingers with the hemiplegic thumb abducted and on top, as in a "prayer position".[8]

Next, the patients are taught how to push their clasped hands forward to assure protraction of the scapula as the arms are lifted above the head. Monitor this movement to assure that the patients are performing it correctly. If carried out improperly, patients may inflict harm to the shoulder or cause pain and be discouraged to move it. If patients are unable to attain full mobility, the family is then instructed to perform the ROM exercises in order to keep the flexibility in the muscles and joints. Insufficient ROM can lead to adhesive capsulitis and shoulder-hand syndrome. Overhead pulleys are contraindicated for self-ROM because they do not provide sufficient scapular mobility with overhead motion of the arm. Kumar et al.[23] evaluated three different groups of upper extremity exercises used in the rehabilitation of stroke patients. The first was ROM exercises performed by a therapist; the second was a figure-of-eight exercise utilizing a skateboard attached to the arm; and finally, the third was overhead pulley exercises. The results of the study showed a significant difference in the development of shoulder pain between the three groups with 8 percent of the subjects in the ROM by the therapist group 12 percent in the skateboard group, and 62 percent in the overhead pulley group developing pain.

Pulling on the arm or letting it hang unsupported during passive transitional movements will increase the risks for a traction injury at the GH joint.[2,11] Slings may assist in supporting the hemiplegic arm during such position changes. Davies[8] and Cailliet[4,11] suggest guiding all movement with the scapula instead of the arm when performing transfers or making positional adjustments. It is very important for physical therapists to educate the hospital staff and family about ways to move stroke patients safely, without pulling on the arms or underneath them.

CHAPTER FIVE

SUPPORTIVE DEVICES FOR THE HEMIPLEGIC SHOULDER

Shoulder subluxation presents a serious problem for the hemiplegic stroke patients and rehabilitation team because it complicates restorative efforts and limits functional recovery.[37] Physical therapists direct much of their attention in providing or maintaining a painfree and functional upper extremity in their patients. They also devote a considerable amount of time in preventing or treating shoulder subluxation resulting from hemiplegia, yet it is this aspect of therapy that great controversy has emerged among health care professionals. Various treatment methods have been devised and advocated for the subluxed shoulder. Of all treatments, the use of slings has probably created the most extensive debates among health care providers.[4] Orthotic aids are constructed to simulate the action of the supraspinatus muscle. During the flaccid stage of stroke recovery, it is believed that slings will provide support for the GH joint and reduce the gravitational load on the joint, thereby preventing inferior subluxation.[21]

Authors have discovered valid reasons to both support and dispute the effectiveness of slings in the prevention and management of shoulder subluxation. Proponents for sling use have suggested that they can help prevent or reduce GH subluxation by counteracting the stresses of gravity on the vulnerable flaccid arm, thereby avoiding the stretch on the joint capsule and ligaments.[14] Others have reported that using slings may essentially reduce the likelihood of developing shoulder pain if subluxation[5] and brachial plexus injuries[37] were avoided. One source cites that using slings, as an "orthosis", may even reduce the occurrence of shoulder hand syndrome.[29]

Opponents have cautioned against the use of slings for several reasons. First, many claim that slings actually contribute to shoulder subluxation rather than prevent it if proper alignment is not maintained.[4,11] Second, slings may reinforce the flexor synergy pattern because they often position the arm in a flexed and internally rotated posture.[5,8,14,38] Third, they may impair balance reactions needed for walking, standing, and sitting since the arm is not free to swing or balance the body. Fourth, they may limit shoulder range of motion by excessively immobilizing the arm, resulting in degenerative changes in the joint and muscle atrophy. Fifth, some patients may feel they interfere with body image. And finally, most supportive devices do not provide adequate motor and sensory stimulation to the hemiplegic limb or allow for bilateral use of the upper extremities.

Some authors note the importance of using slings for upper limb support, but they often advise that their usage be limited in order to avoid the complications listed above. Some clinicians suggest that patients be allowed to wear slings only during ambulation, while others recommend wearing the slings in sitting as well as during gait activities.[31] Patients with severe unilateral neglect or sensory and attentional deficits may need a sling to protect the arm from trauma. Patients suffering from right cerebral damage appear to show hemineglect more frequently and to a greater extent than patients with left cerebral damage. Hemiplegic patients with neglect may be at an increased risk for shoulder trauma since they often demonstrate careless and impulsive behaviors. In one study, it was hypothesized that hemineglect may be a precipitating factor in producing hemiplegic shoulder pain.[9] However, the investigators could not gather significant evidence to establish a relationship between unilateral neglect and shoulder pain, even though right CVA patients did encounter shoulder pain more frequently. Furthermore, the patients with shoulder pain also had more spasticity, decreased sensation, and shoulder subluxation.

A magnitude of sling designs are available for application. They all have their advantages and disadvantages. Some are easy to apply and allow for independent donning while others are very complex, requiring the assistance of family or medical staff for application. Some allow for free arm movement and bilateral use of hands while others restrain the arm against the body. Many fabrications have been done to improve the design of the slings for better acceptance and functional use. Unfortunately, few studies have used radiographic evidence to compare the effects of different types of slings on reducing shoulder subluxation.[16,31] In one efficacy study, Hurd and colleagues found the hemisling to be ineffective in reducing shoulder subluxation and pain in their subjects.[39] No appreciable differences in the degree of subluxation were found between the experimental group who wore the slings and the control group who did not. In a more recent study, Brooke et al.[16] recorded that the Harris hemisling was effective in reducing both vertical and horizontal GH displacement. The vertical component of GH joint alignment was found by measuring the distance between the central point of the humeral head to the acromial point. The horizontal component was determined by measuring the central points between the humeral head and glenoid fossa.

Since comparative investigations are sparse, physical therapists must rely on their clinical experience and judgment to determine the effectiveness of a shoulder support system. When considering the utilization of a sling, attention must also be given to patient acceptance, comfort, ease of application, and cost.[39]

Besides slings, other supportive devices have been used to manage the hemiplegic upper extremity. These include padded arm troughs and lap trays attached to the arm rests of the wheelchair and overhead wheelchair slings. It is still questionable which method is more effective in preventing or reducing GH subluxation because no studies were found in the literature that compared these three. However, studies have been done using the arm trough and lap tray as supportive devices. An early study conducted by Moodie et al.[21] compared the effects of the arm trough and lap tray along with three other slings in reducing an already existing shoulder subluxation. They found the wheelchair supports to be effective in reducing shoulder subluxation, although, the arm trough was slightly superior to the lap tray. In a more recent investigation, researchers found the lap tray and arm trough to be less effective than their counterpart, the Harris hemisling, in reducing vertical subluxation.[16] In fact, the wheelchair attachments tended to overcorrect the subluxation upon X-ray measurements.

Despite the consequences documented in the literature on supportive aids, orthotic management of GH subluxation is considered to be an important component of the comprehensive rehabilitation program, especially during the flaccid stage of recovery. For example, the Harris hemisling provided good correction and yielded results that were consistent.[16] Although the arm trough and lap tray are less effective in reducing subluxation, they allow the arm to be positioned out of the flexor synergy pattern and permit more freedom to move.

CONVENTIONAL SLING

The conventional sling[21] is easy to assemble. It is made of cotton fabric and is triangular in shape. To wear this type of sling, the elbow must be flexed to 90 degrees in order to properly support the forearm in the horizontal plane. The ends of the sling are fastened behind the neck.(Fig. 4) This type of support is desirable for patients who have edema in the wrist and hand or have pain in the shoulder region with the arm in a dependent position.

In one particular study, the conventional sling reduced shoulder subluxation to within 20 percent of correct alignment on anterior-posterior radiographs in 8 out of 10 subjects studied.[21] It proved to be the most effective aid out of the five studied in managing GH subluxation. In spite of its good support, the researchers cautioned against its use. Because the conventional sling positions the patient in an undesirable flexor synergy pattern, they feel it should only be worn if a painful joint is interfering with therapy and prohibiting function. It might also be helpful in temporarily supporting and

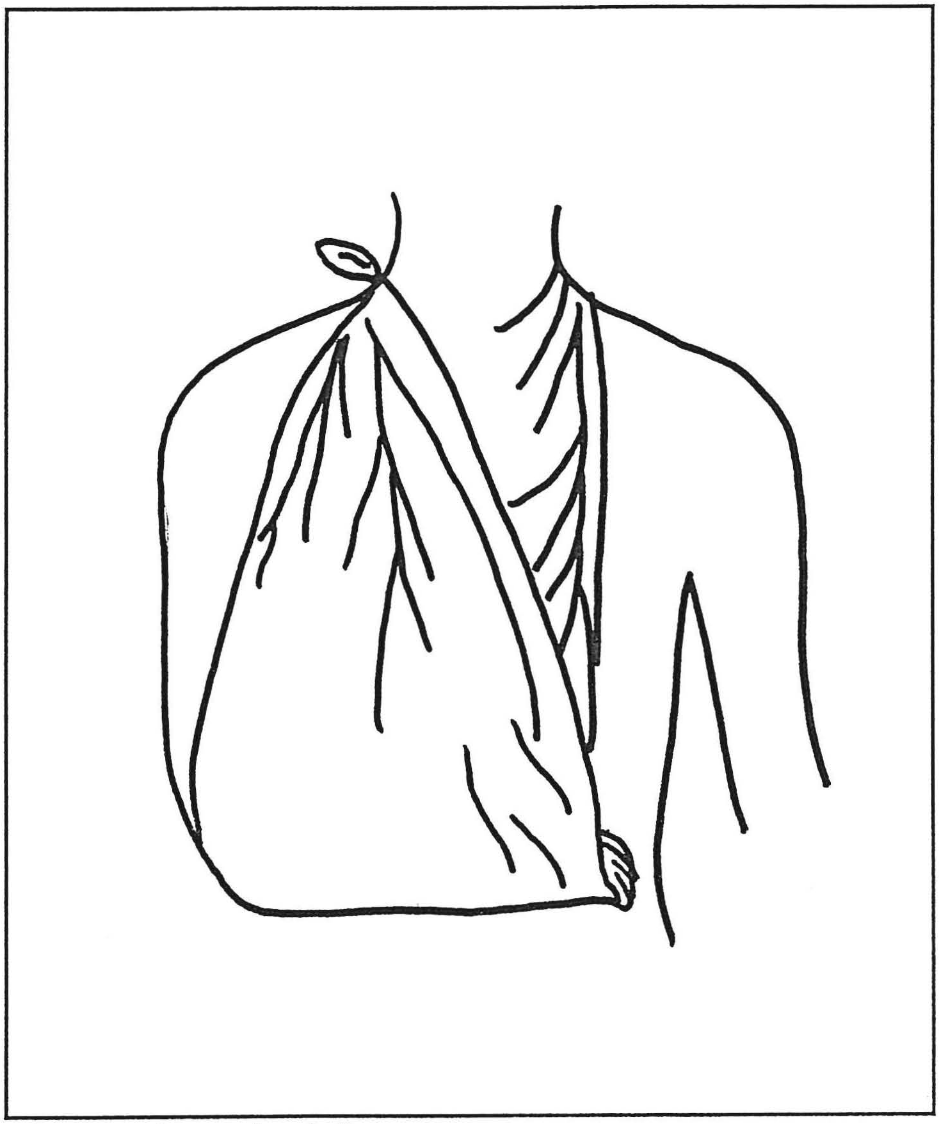


Figure 4. Conventional sling.

protecting the upper extremity when learning new skills, such as transfers and gait training.

BOBATH SHOULDER ROLL

This aid attracted the interests of many clinicians when first introduced by Bobath in 1978, because it allowed the extremity to remain in an extended rather than flexed position.[38] Since then it has been included in several research studies. The shoulder device consists of three main parts:[21] 1) a foam roll placed in the axillary region of the affected shoulder, 2) a vertical strap made of cotton webbing that secures the roll in place, and 3) a horizontal strap made of similar material that encircles around the chest and attaches to the vertical strap via D-ring and Velcro closure. (Fig. 5)

Moodie et al.[21] found the Bobath roll to be ineffective in reducing shoulder subluxation in their subjects. Brooke and associates[16] made some minor adjustments to the Bobath roll and included it in their study. Instead of a horizontal strap, it contained a figure 8 pattern that connected between the shoulder blades. (Fig. 6) With the alterations, the sling did provide some vertical correction, although it was not as significant as the other device studied, which was the Harris hemisling. Another important finding was that the sling contributed to the horizontal displacement of the GH joint due to the shoulder roll placed in the axilla. They concluded that this type of device may be appropriate for patients with mild subluxations complicated by spasticity since it positions the arm out of the flexor synergy pattern. On the other hand, it would be inappropriate for patients who

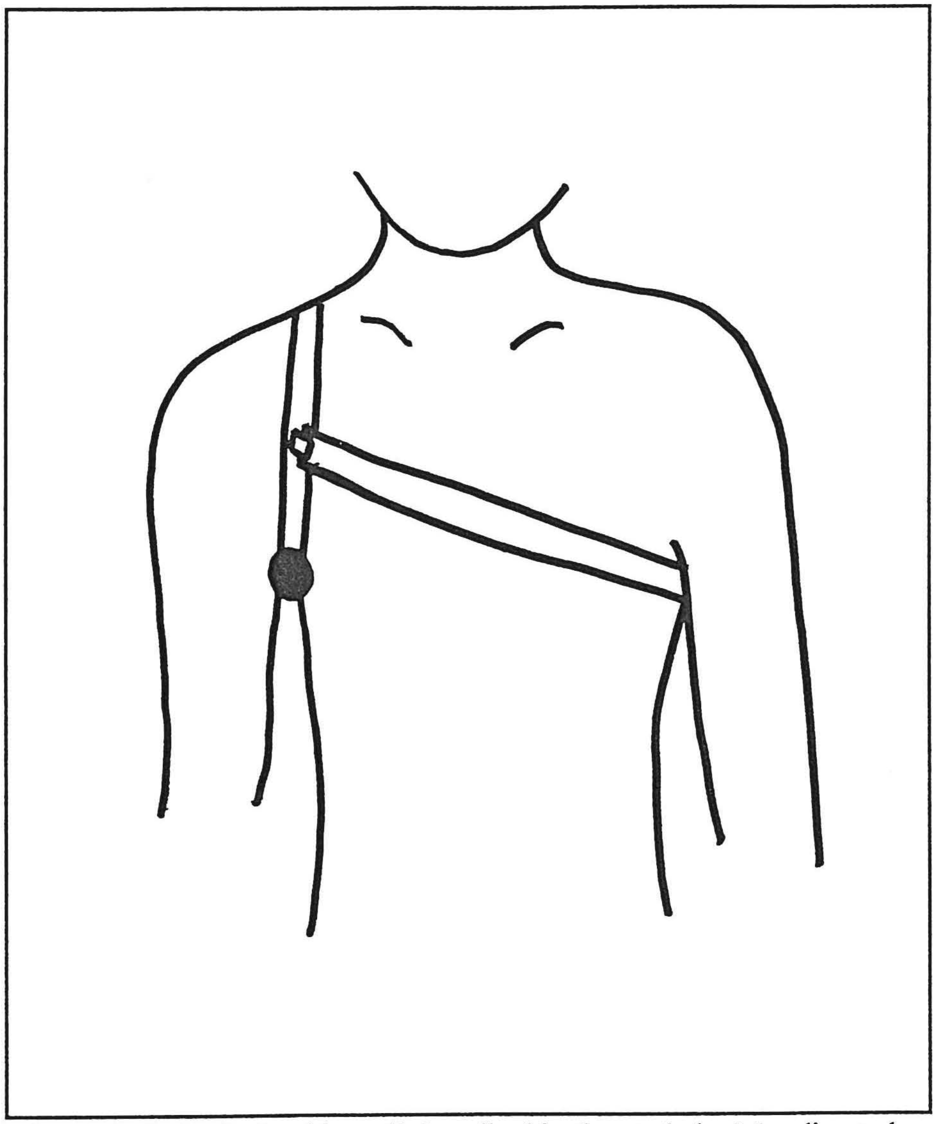


Figure 5. Bobath shoulder roll described in the study by Moodie et al.

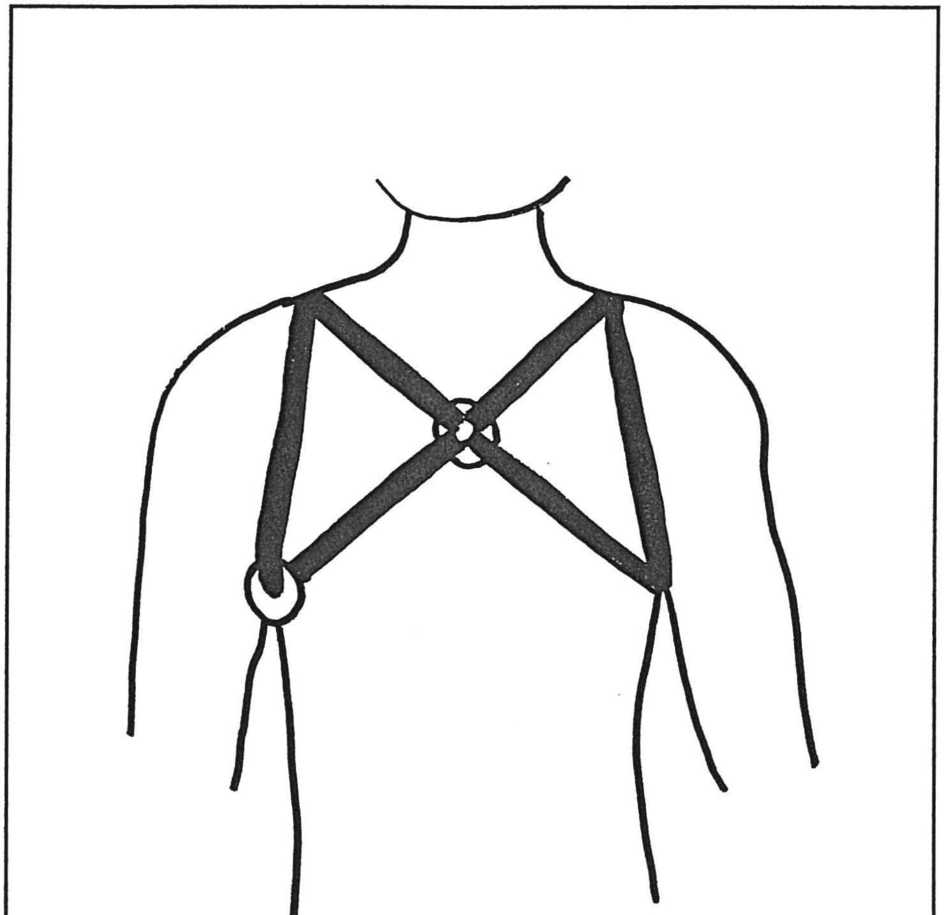


Figure 6. Bobath shoulder roll described in the study by Brooke et al.

have moderate to severe subluxations since it failed to significantly reduce the vertical displacement.

Advantages of the Bobath sling are that it allows free arm movement and bilateral use of the extremities. It provides constant upper extremity input since the patient is able to do weigh-bearing activities with the sling on.[38] The disadvantages of the sling are that it laterally displaces the humerus into abduction; it is difficult to don and doff independently; and it lacks distal support, therefore it can lead to increased hand edema or trauma. Careful monitoring of circulation and sensation are necessary when using this type of device so that compression of the brachial plexus and axillary artery do not occur from the axilla roll.

HARRIS HEMISLING

The Harris sling[16] consists of an elbow enclosing pad, a second pad under the wrist and hand, and adjustable loops extending from the pads to a connecting piece in back. (Fig. 7) Brooke et al. discovered from their measurements that the sling provided good correction in both vertical and horizontal directions on anterior-posterior radiographs. A negative consequence of this sling is that it may promote the flexor synergy pattern since it positions the shoulder in adduction and internal rotation.

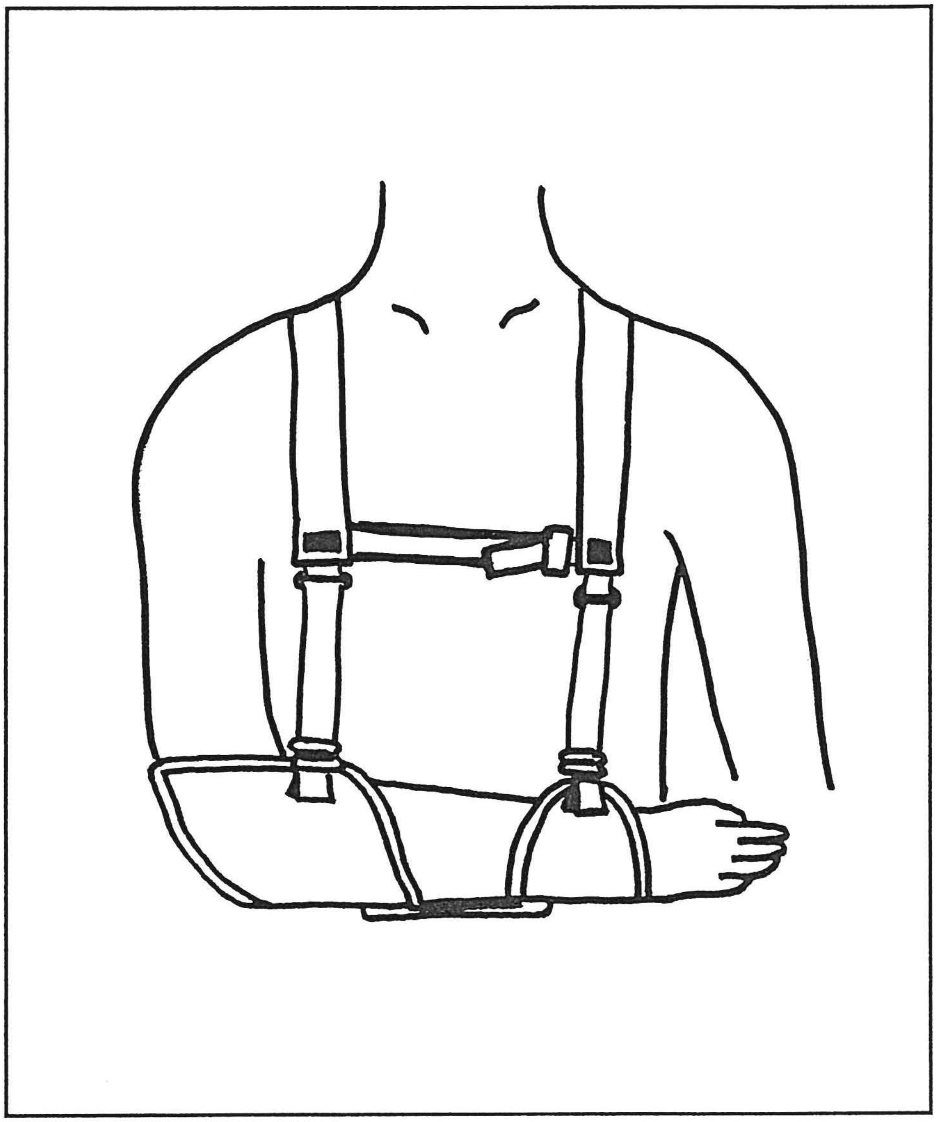


Figure 7. Harris hemisling.

ROOD SLING AND MODIFIED ROOD SLING

The composition of the Rood sling[4,11] is quite different than the others discussed so far, in that it consists of elastic tubing and a cone-shaped device for the hand. (Fig. 8) The elastic tubing provides dynamic or kinetic support and stimulates extension of the arm. The cone spreads the fingers and thumb and radially deviates the wrist. Proper application allows the forearm to be supinated and the scapula to be elevated and derotated. This device offers proprioceptive stimulation by forcing the humeral head up into the suprahumeral joint space.

Components of the Rood Sling attracted the interests of several physical therapists and an occupational therapist and it led to the formation of the Modified Rood Sling.[6] (Fig.9) Its purpose was to decrease GH subluxation and pain in the flaccid shoulder when upright and out of the wheelchair. It was comprised of a foam axilla pad, an elbow cuff, a cone for the hand, and elastic tubing for the straps. Adler-Traines noted that all patients with flaccid upper extremities, who were ranged without scapular mobilization and/or positioned in slings that promoted flexion and internal rotation, developed GH subluxation and pain. Meanwhile, subluxation and pain were either prevented or decreased in 100 percent of the patients when the modified Rood sling and overhead suspension sling were applied and scapular mobilization was performed during exercises. Besides decreasing the subluxation, other benefits noted with this sling are that it facilitates the surrounding shoulder musculature and increases patient comfort. Unfortunately, patients, staff, and family found the Modified Rood Sling hard to apply due to its complex design.

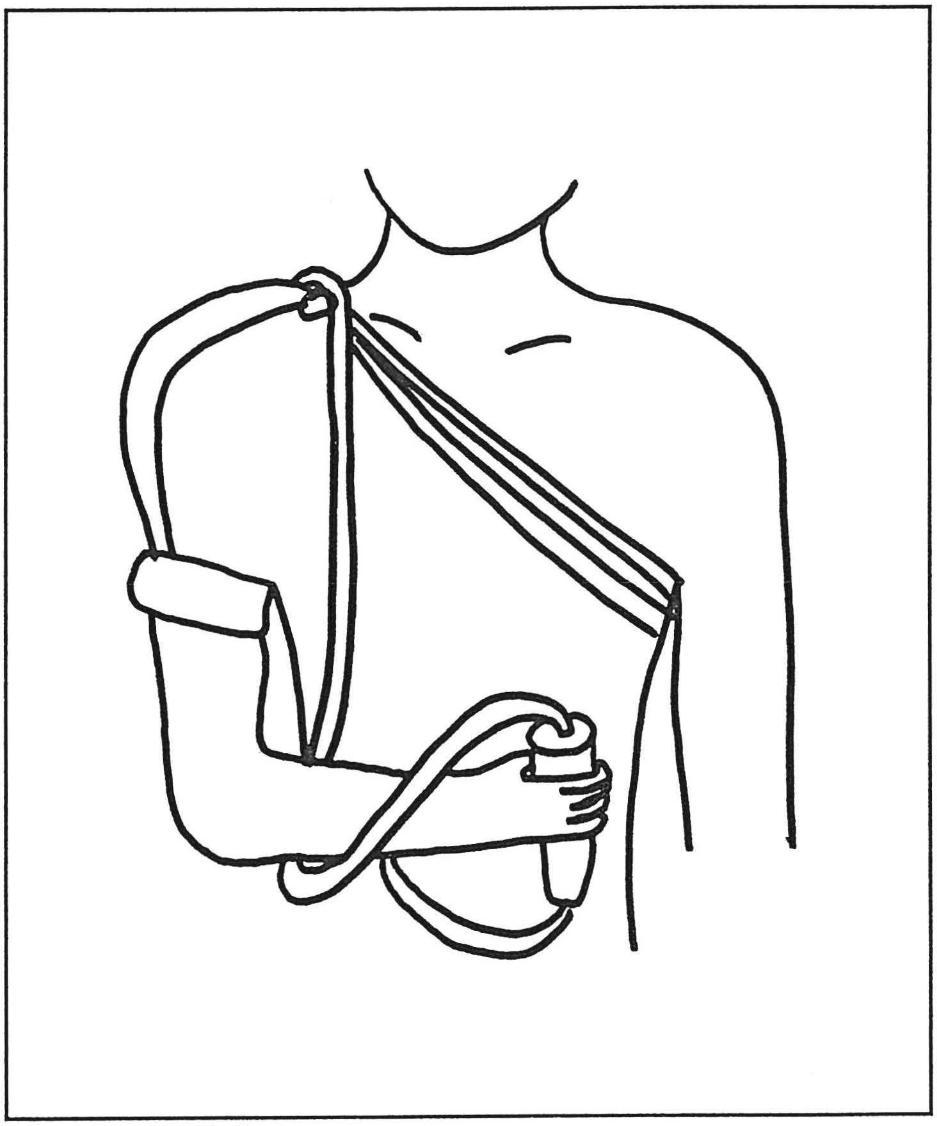


Figure 8. Rood sling.

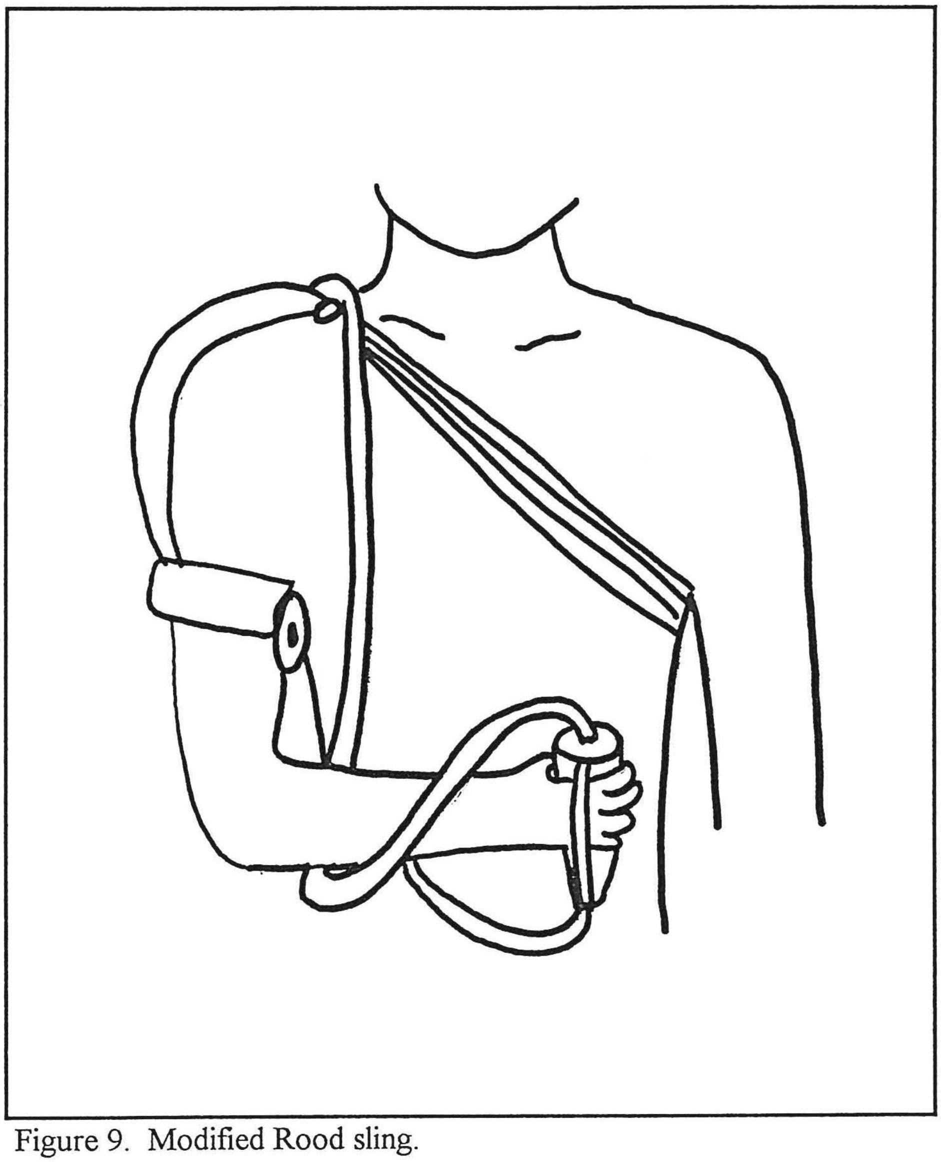


Figure 9. Modified Rood sling.

WHEELCHAIR ARM SLING

Overhead slings work well for the patient who is confined to the wheel chair and need the arm support of a sling.[4,11] (Fig. 10) This type of device aids in controlling edema. It holds the shoulder in a forward flexed position with the elbow, wrists, and fingers extended. It also minimizes shoulder subluxations and prevents disabling deformities while permitting full arm motion.

KOHLMEYER-RIC ORTHOSIS

The Kohlmeyer-RIC[37] orthosis was initially designed for patients with central cord syndrome and brachial plexus injuries.(Fig. 11) Fabrication of this brace takes only 2-4 hours and it uses readily available materials. Two patients with extension spinal cord injuries and one patient with bilateral brachial plexus injuries, resulting from an industrial accident, benefited from this type of device. Before application, patients were dependent in ADLs, received muscle grade tests ranging from 0 to 5 for the shoulder girdle and upper extremity musculature, demonstrated shoulder subluxation, and complained of pain. The orthosis allowed the patients to be independent in most all ADLs by using trunk flexion movements and adjusting the degree of arm position. Pain complaints decreased substantially when wearing the brace and increased when not wearing it. Shoulder subluxation was also reduced with the orthosis on. Lastly, it improved arm swing and balance during gait. This brace has shown to be cost effective for these patients when

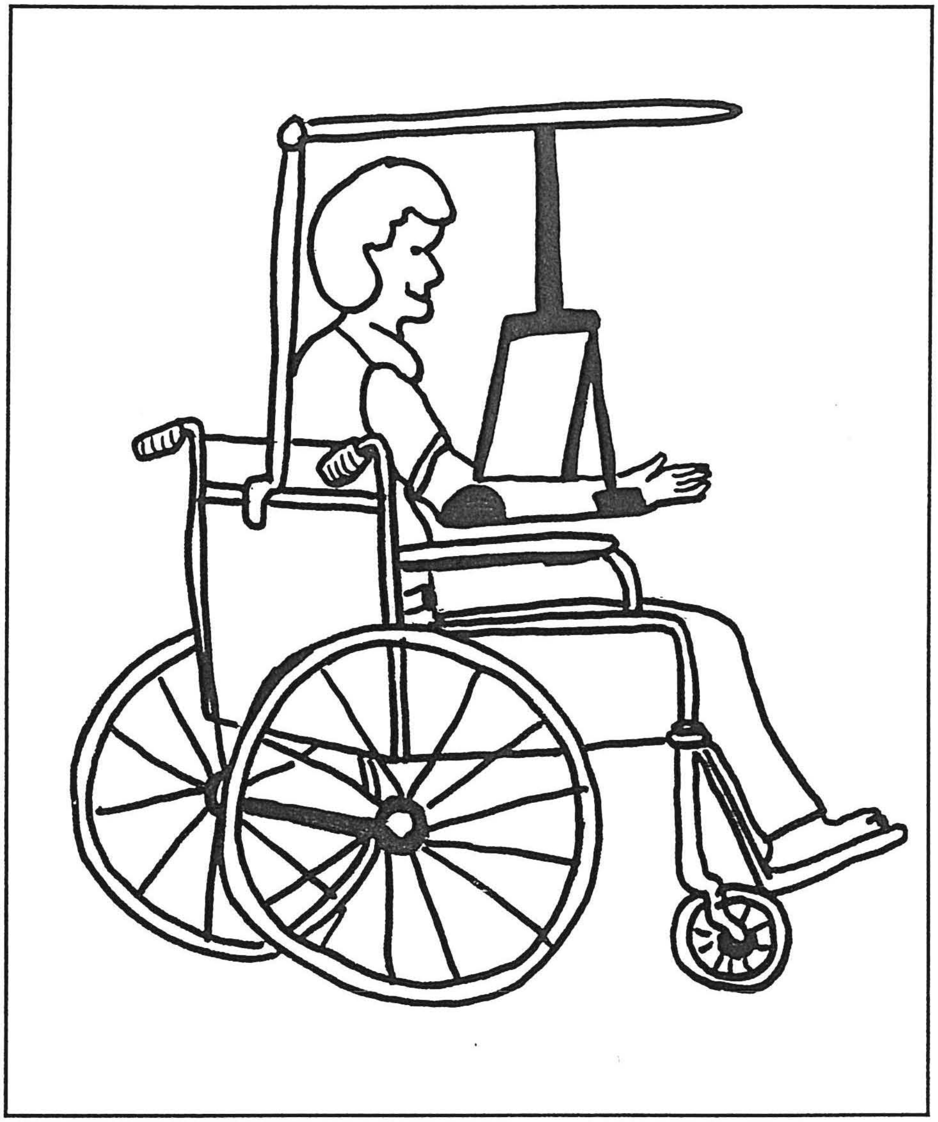


Figure 10. Wheelchair arm sling.

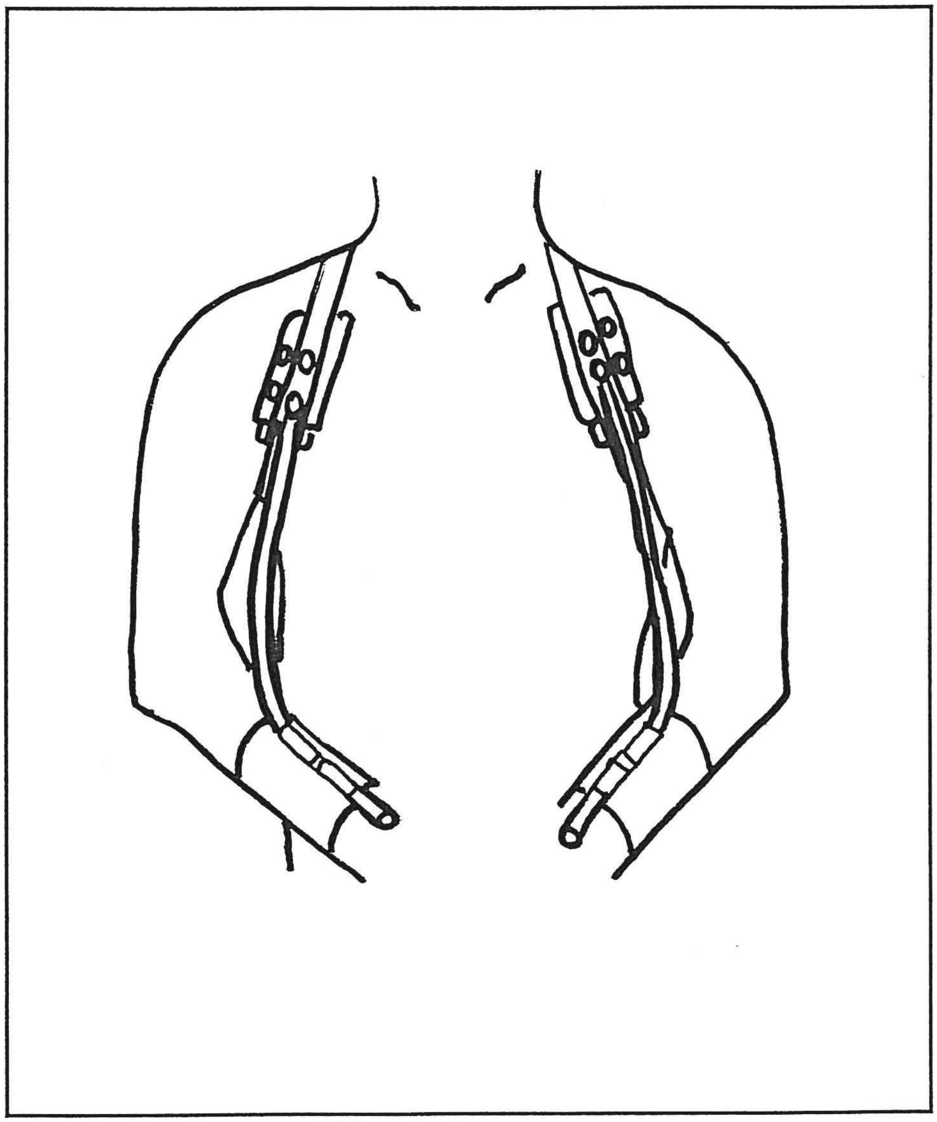


Figure 11. Kohlmeyer-RIC orthosis.

motor return is possible. This type of device may be applicable for the stroke patients who demonstrate a loss of motor function, especially in the proximal musculature. It has the added benefits of being lightweight and avoiding abnormal synergy postures.

ARM TROUGH AND LAP TRAY

The arm trough (Fig. 12) and lap tray (Fig. 13) attach to the armrests of the wheelchair. Clinically, arm troughs hinder bilateral use of the upper extremities which may contribute to the complications associated with immobilization and decreased sensory awareness.[21] Lap trays allow for greater freedom and allow for bilateral use of the hands. A disadvantage of both of them is that the shoulder position is altered when the patient changes positions in the chair, either intentionally during pressure relief exercises or unintentionally with a cough, sneeze, or slouching in the chair due to poor trunk stability.[16] The height of the patient in sitting, the length of the upper limb, and the height of the arm rest and assistive device must be accounted for so that the pressure at the elbow is not forcing the humeral head up into the coracoacromial arch.

Figure 12. Wheelchair arm trough.

Figure 13. Wheelchair lap tray.

CHAPTER SIX

CONCLUSION

The hemiplegic shoulder has been extensively studied over the years to search for effective prevention and treatment strategies of post-stroke complications. A treatment technique that is completely successful in managing shoulder dysfunction has yet to be discovered. The shoulder will continue to receive much attention in the future because it appears to be an ongoing problem within the hemiplegic stroke population.

Glenohumeral subluxation and pain are the primary shoulder complications found in the literature. Rotator cuff tears, impingement disorders, and brachial plexus injuries have also been identified in stroke survivors. These complications may or may not be associated with discomfort. A painful shoulder is likely to become a major barrier in the rehabilitation process. It may lead to uncooperative behaviors, depression, decreased motivation, and impaired function. As a result, the functional prognosis of the paralyzed limb may be greatly reduced.

There appears to be an increased risk of developing shoulder complications in the flaccid phase of stroke recovery. The cause for this is uncertain; however, experts speculate that the shoulder is unstable during this stage and becomes susceptible to injury as the rotator cuff mechanism experiences an abnormal change in muscle tone. Shoulder subluxation occurs when the head of the humerus separates from the glenoid cavity of the scapula. The subluxation does not usually produce pain initially, but it may become painful if distracting forces cause persistent malalignment. Patients with GH subluxation may never develop shoulder pain if the scapula is kept mobile on the body.

Subluxation and pain often occur simultaneously. It is not certain whether the subluxation is the contributing factor in producing the pain, but it is presumed that periarticular overstretching causes the receptors within the coracohumeral ligament to elicit painful responses.[20] It has also been speculated that constant distractive forces may be responsible in producing tendon ischemia in the supraspinatus and biceps brachii musculature. There is reason to believe that this ischemic condition is what causes the shoulder pain in so many stroke patients. Shoulder subluxation does not always produce pain symptoms and shoulder pathologies other than subluxation may be instrumental in causing the intense pain.

A significant relationship does not appear to exist between the degree of pain and severity of subluxation.[20] A higher grade of GH subluxation is not associated with greater tissue damage and pain. The extent of damage may depend on how long and how often the arm is left unsupported, and not on the severity of the subluxation.

Shoulder-hand syndrome or reflex sympathetic dystrophy is a debilitating complication that patients may encounter 1-6 months after the onset of stroke. The cause of this disorder has yet to be identified as well. Immobilization of the shoulder, prolonged flexion of the wrist, recurrent damage to the wrist and hand, disturbances of the vasomotor regulatory system, or lesions of the premotor and motor cortex may trigger the syndrome. It is characterized by pain, stiffness, edema, deformities, and trophic skin changes of varying degrees. Both the hand and shoulder may be involved. It seems to be associated with patients who have greater subluxations, have more confusion, have more sensory disturbances, or have a history of myocardial infarction.[31]

A multitude of treatment regimens for the hemiplegic shoulder have been advocated. Before management can begin, one must demonstrate a thorough understanding of the intricate shoulder complex in conjunction with the problems that may result from central nervous system damage. Most post-stroke shoulder complications are preventable and can be effectively treated when recognized.[7]

Anticipation and prevention of shoulder complications are key components in the rehabilitation of stroke patients. Most experts agree that early mobility, positioning, and safe handling are critical first steps toward the prevention of possible complications. Physical therapists have many responsibilities to insure that the integrity of the shoulder is maintained throughout the entire day. Educating the staff involved with direct patient care is of primary importance.[7] Scheduling regular inservices, displaying signs in the patients' rooms, and enforcing documentation on patient contacts are helpful ways in which therapists can inform and monitor the care given by others. Every team member must become an active part in the rehabilitation program to provide the highest level of quality care in the most cost-effective manner.

Patient and family education is another important aspect of therapy. Rehabilitation is a life-long commitment for persons who have sustained motor and sensory deficits from central nervous system damage. Instruction in exercise and positioning programs and promoting safety awareness will help to maintain the optimal level of upper extremity function. Without proper exercise or support, the shoulder is subject to complications associated with immobilization or improper positioning.

During the flaccid stage of stroke recovery, experts recommend positioning the upper extremity out of the typical synergy patterns to discourage the onset of post-stroke spasticity. The scapula should be protracted and the upper extremity brought forward and away from the body while engaging in passive pursuits in the bed or chair. A regular turning schedule that incorporates all positions is advised at least every two hours to avoid the development of secondary complications. Lying on the hemiplegic side should be enforced as soon as possible. It reduces spasticty formation and provides stimulation to the affected side through weight-bearing forces. The supine position is indicated for patients who have respiratory complications or for those who do not tolerate the sidelying position. If other positions are tolerated, the time spent in supine should be limited, as it increases the risk of pressure ulceration and abnormal reflex or extensor activity.

The upper extremity must be handled appropriately during ROM exercises and functional activities to minimize the risk of subluxation, impingement, and/or brachial plexus injuries. Mobilizing the scapula and providing adequate rotation of the humerus with arm elevation over the head is important. Exercise methods that do not provide for these motions should be avoided.

Often times, stroke patients need assistance with transfers and positioning. Rather than pulling on the patient's arm or holding underneath the shoulders, shifting the patient's body weight forward and guiding the movement with the scapula is advised. This is another crucial area in which physical therapists need to educate all those involved with patient care.

The use of upper extremity supportive aids is still a controversial issue. The rationale for using supportive devices is to protect the flaccid shoulder from distractive forces during functional activities. Reasons to both support and criticize the use of slings are well documented in the literature.

The Harris hemisling and conventional sling appear to be the most effective in reducing GH subluxation and/or pain. The disadvantage of these slings is that they position the arm in an undesirable posture of shoulder internal rotation and elbow flexion. The Modified Rood sling also proved to be effective in reducing shoulder complications when used in conjunction with overhead suspension devices and scapular mobilization during ROM exercises. This sling has the capabilities of facilitating the shoulder musculature and positioning the arm out of the flexor synergy pattern. The Bobath sling has been indicated for patients with mild subluxations and those who have an increase in tone since it allows for free arm movements and bilateral use of the hands. The Kohlmeyer-RIC orthosis has promoted functional use of the arms and has reduced shoulder subluxations in patients with spinal cord damage or brachial plexus injury. It may be helpful for stroke patients who demonstrate paralysis, especially of the proximal shoulder musculature. The arm trough and lap trays are commonly used to support the upper extremity while in a wheelchair. These devices have been found to be effective in reducing subluxation but special attention must be given to the position of the elbow and forearm.

Future work in this area of stroke management must be done to eliminate some of the uncertainties created by shoulder complications and treatment. Future investigations into this problem are warrant since hemiplegic complications present such major obstacles in the rehabilitation of stroke patients. More knowledge is needed concerning the source and course of development of shoulder dysfunction after stroke. Deficits in perception and sensation may play a role in the pathogenesis of shoulder pain and dysfunction, which may be the reason why persons with left hemiplegia experience more problems than right hemiplegia. However, the role of perceptual abnormalities in the development of pain requires more detailed study.[9,19] There are various types of supportive aids available on the market but there have been a limited number of studies performed on comparing the effectiveness of them. Prospective controlled research is needed to establish a causal relationship between the type of support and clinical signs and symptoms.[16]

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